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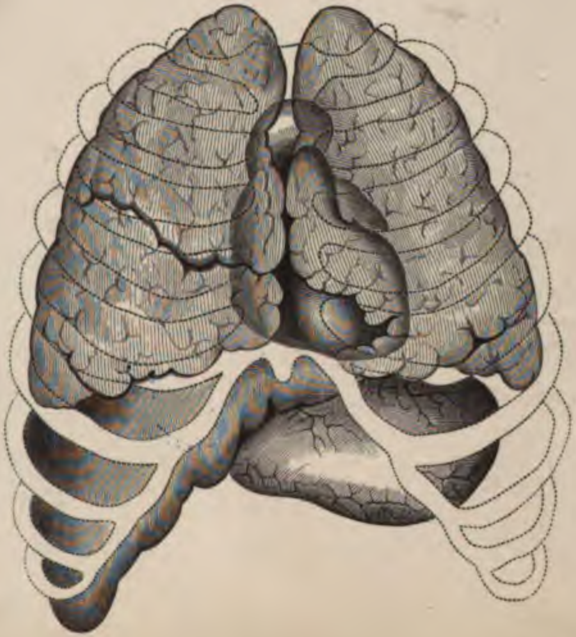

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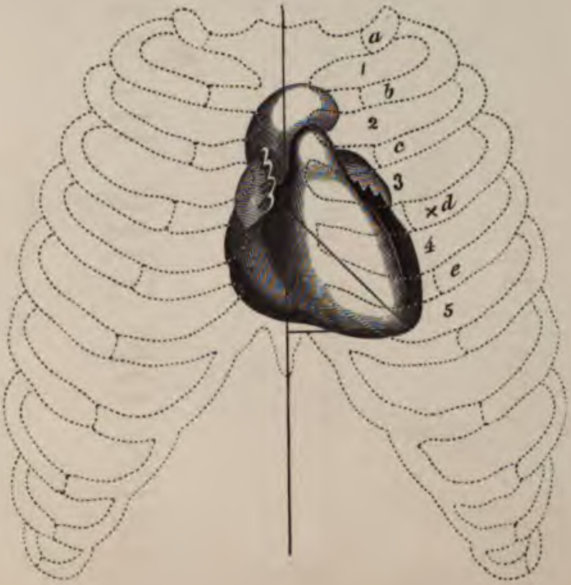

For Description, see page xv.

Chas. M. Allin

A PRACTICAL TREATISE

ON THE

DIAGNOSIS, PATHOLOGY, AND TREATMENT

OF

DISEASES OF THE HEART.

BY

AUSTIN FLINT, M.D.,

PROFESSOR OF THE PRINCIPLES AND PRACTICE OF MEDICINE, AND OF CLINICAL MEDICINE,
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PREFACE TO THE SECOND EDITION.

In revising this treatise for the second edition, the author has endeavored to incorporate the results of the study of the diseases of the heart during the ten years which have elapsed since the appearance of the work. Many additions and alterations have been made, and much has been rewritten. An analysis of about four hundred and fifty cases, recorded by the author during the period just stated, has served as the basis of the revision.

March, 1870.

PREFACE TO THE FIRST EDITION.

In the preparation of this volume, the aim has been to meet the wants of the medical student and practitioner by the production of a work devoted exclusively to diseases of the heart, and treating concisely, but comprehensively, of these diseases with reference to their diagnosis, pathology, and treatment. Such a work, if satisfactorily executed, it is believed, can hardly fail to prove acceptable, in view of the importance of this class of diseases, the progress made in their investigation during the last few years, and the absence of any extended text-book, published in this country, having the same scope and objects, since the appearance of Dr. Hope's treatise twenty years ago. The need of a practical work on diseases of the heart is so apparent, that the present effort requires no apology ; and if not successful, the fault must be imputed to the performance rather than to the undertaking. The author ventures to hope, in submitting this volume to the profession, that it may be found, in some measure at least, to supply a desideratum, the existence of which must have been felt by many practising physicians, and, more especially, by medical teachers and their pupils.

It will be observed that the arrangement of subjects in this work differs from that generally adopted. As regards the order in which the different diseases are considered, the plan usually pursued may be said to be synthetical, inflammatory affections being taken up first, and afterward the lesions which are, to a considerable extent, results of inflammation. A method which may be distinguished as analytical, has appeared to the author preferable. Pursuing this method, the work commences with the consideration of organic affections. Enlargement of the heart, occurring often consecutively to other lesions, takes precedence. To this subject the first chapter is devoted. Lesions affecting the walls of the heart naturally come next in order. These constitute the subject of the second chapter. Valvular lesions are then considered, occupying two chapters, and a chapter is devoted to congenital malformations. Several affections which are incidental to diseases of the heart, are treated of in a distinct chapter. Then follow the inflammatory affections, and, afterward, functional disorder of the heart, three chapters being allotted to these classes of disease. Finally, thoracic aneurisms, which claim consideration in connection with diseases of the heart, are made the subject of the concluding chapter.

In writing the book, the end which the author has kept steadily in view is, a fair and full exposition of our present knowledge of the diagnosis, pathology and treatment of diseases of the heart. Recognizing clinical study as the great source of this knowledge, he has endeavored to make the cases reported by trustworthy observers, together with his own recorded experience, the basis of the work. Having long been in the habit of making records at the bedside, and having given for several years particular attention to diseases of the heart, he has accumulated notes of about two hundred cases of the various cardiac affections. The results of an analysis of these cases have been before him during the composition of the work. As a preliminary step, also, over one hundred fatal cases gathered from different authors, chiefly from the works of Hope, Stokes, Andry, and Blakiston, were subjected to similar analysis. On the data thus obtained have been based, in a great measure, the statements and opinions which the work contains, endeavoring, however, not to introduce details and statistics to an extent to prove repulsive or fatiguing to the reader. But although it may be claimed in behalf of the work that it is something more than a compilation, not to have studied closely the literature of the subject would have been an injustice alike to it and to those by whose labors this department of practical medicine owes its present development. Of the authors to whom acknowledgments are due, the names of Bouillaud, Hope, Stokes, Walshe, Andry, Forget, and Bellingham, are to be especially mentioned. References to these and others will frequently occur in the following pages. The author has aimed to prepare a practical treatise, and he has therefore avoided, or dismissed with as much brevity as possible, speculative opinions and mooted questions involving discussions which would occupy space to the disparagement of matters relating more directly to medical practice. It may seem, nevertheless, to some, that the volume is out of proportion to the field of practical medicine to which it is restricted; but it is hoped there will be no reason to complain of a redundancy either in style or matter, and that the reader will be led to attribute the size of the book to the progress of knowledge pertaining to diseases of the heart, together with their intrinsic claims on the attention of the student and practitioner.

A liberal share of the work is devoted to physical signs. But a just estimate of their practical importance will obviate any objection on this score. It is mainly owing to physical exploration that the study of these diseases has been prosecuted within the past few years with such remarkable success. Here, as in other classes of affections, the knowledge to be derived from clinical observation is increased in proportion to improvement in diagnosis, and it is evident that diseases cannot be judiciously treated unless correctly discriminated. The discrimination of diseases is confessedly the portion of our art which involves the most difficulty and calls for the greatest amount of skill. Hence, it is especially under this practical aspect that diseases in general claim careful and extended consideration. This remark, certainly, is not less applicable to diseases of the heart than to other nosological divisions. And the diagnosis of cardiac diseases is for the most part based on the physical signs. It is, therefore, by no means solely because these are interesting, but on account of their practical importance, that so much space has been accorded to them in the present treatise. In treating of the physical signs, it was necessary to introduce some matter belonging properly to anatomy and physiology, viz., the relations of the heart to the walls of the chest and the adjacent viscera, the movements of the organ, and the normal heart-sounds. With reference to the movements and sounds of the heart, the author has been led by examinations of the healthy chest to conclusions which appear to have important practical bearings. The abnormal modifications of the heart-sounds have hitherto scarcely received sufficient attention. More importance is attached to them as diagnostic signs, and they are considered more fully in this work than in any other on the diseases of the heart with which the author is acquainted. As regards the sounds of the heart in health and disease, some original views are introduced, which have entered into a previous publication.[1]

In thus setting forth, briefly, the plan and objects of the work, the author assumes only to have spared no pains to render it acceptable to the profession. All who have engaged in similar undertakings amidst the cares and distractions of active medical practice, will appreciate the difficulty of the task. But the time and labor which the author has bestowed upon it, will be more than requited by the approval of his medical brethren; and he is encouraged to hope for this reward by the favor with which his previous contributions to practical medicine have been received.

The author would express his thanks to Prof. John C. Dalton, Jr., for the two illustrations which form the frontispiece, and for other friendly offices; also, to Dr. Austin W. Nichols, formerly assistant to the chair of clinical medicine in the University of Buffalo, for his valuable assistance in collecting materials for the preparation of the work.

NEW YORK, September, 1859.

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[1] On the Clinical Study of the Heart-Sounds in Disease and Health. Transactions of the American Medical Association for 1858.

CONTENTS.

CHAPTER I.

ENLARGEMENT OF THE HEART.

PAGE

Definition and varieties of hypertrophy and dilatation—Normal dimensions and weight of the heart—Enlargement by hypertrophy—Concentric hypertrophy—Symptoms and pathological effects of hypertrophy—Physical signs and diagnosis of enlargement and hypertrophy—Situation and anatomical relations of the heart in health—Alterations in degree and extent of dulness on percussion in hypertrophy—Altered situation and extent of the apex-beat, and abnormal force of impulse in hypertrophy, as determined by palpation — Abnormal modifications of the heart-sounds—Diminished extent and degree of the respiratory murmur and vocal resonance within the præcordia in hypertrophy, as determined by auscultation—Results of the clinical study of the heart-sounds in health —Enlargement of the præcordia and abnormal movements in hypertrophy, as determined by inspection—Increased size of the chest, as determined by mensuration—Summary of the physical signs of enlargement of the heart—Summary of the physical signs distinctive of enlargement by hypertrophy—Treatment of hypertrophy—Enlargement by dilatation—Symptoms and pathological effects of dilatation—Physical signs and diagnosis of dilatation—Summary of the physical signs distinctive of enlargement by dilatation—Treatment of dilatation, . . 17

CHAPTER II.

LESIONS, EXCLUSIVE OF ENLARGEMENT, AFFECTING THE WALLS OF THE HEART.

Atrophy of the heart—Fatty growth and degeneration—Symptoms and pathological effects of fatty growth and degeneration—Physical signs and diagnosis of fatty growth and degeneration—Treatment of fatty growth and degeneration—Softening of the heart in typhus and typhoid fever and other affections—Treatment of softening of the heart—Induration of the heart—Cardiac aneurism and aneurism of the coronary artery—Rupture of the heart—Carcinoma, tuberculosis, extravasation of blood and cysts, ... 91

CHAPTER III.

LESIONS AFFECTING THE VALVES AND ORIFICES OF THE HEART.

PAG

Aortic lesions—Mitral lesions—Primary effects of valvular lesions on the circulation—Points to be observed in post-mortem examinations—Pathological processes involved in the production of valvular lesions—Symptoms and secondary pathological effects of lesions affecting the valves and orifices of the heart—Symptoms and pathological effects referable to the heart—Enlargement of the several portions of the heart in relation to mitral, aortic, tricuspid, and pulmonic lesions, respectively—Pain, palpitation, the pulse, venous turgescence, and pulsation—Symptoms and pathological effects referable to the circulation—Cardiac dropsy—Arterial obstruction by emboli—Symptoms and pathological effects referable to the respiratory system: Dyspnœa, cardiac asthma, cough, muco-serous expectoration, hæmoptysis, pulmonary apoplexy, œdema, bronchitis, pneumonitis, pleurisy, and emphysema — Symptoms and pathological effects referable to the nervous system: Apoplexy, paralysis, arterial obstruction, defective supply of blood to brain, pseudo-apoplexy, cephalalgia, vertigo, tinnitus aurium, &c., sleep, mental condition—Symptoms and pathological effects referable to the digestive system and nutrition: Hepatic congestion, nutmeg liver, portal congestion, enlargement of liver, cirrhosis, indigestion, gastrorrhagia, enterorrhœa, melæna, hemorrhoids, enlargement of spleen, nutrition—Symptoms and pathological effects referable to the genito-urinary system: Congestion of kidneys, diminished secretion of urine, albuminuria, structural degeneration of kidney or Bright's diseases—Generative functions—Symptoms and pathological effects referable to the countenance and external appearance of the body: Lividity, expression, anæmia, capillary congestion, erythema, bloodless fingers, ... 12

CHAPTER IV.

PHYSICAL SIGNS, DIAGNOSIS, AND TREATMENT OF VALVULAR LESIONS

Endocardial or valvular murmurs—Distinction between endocardial and exocardial murmurs—Vascular murmurs — Distinction between inorganic and organic murmurs—Soft and rough murmurs—Musical murmurs—Enumeration of abnormal conditions giving rise to endocardial murmurs, organic and inorganic—Valvular lesions involving obstruction or regurgitation, or both, generally accompanied by a murmur—Circumstances enabling the auscultator to determine whether lesions involve obstruction, or regurgitation, or both—Mitral direct, or systolic murmur—Mitral regurgitant or diastolic murmur—Aortic direct or systolic murmur—Aortic regurgitant or diastolic murmur—Localization of systolic murmurs—Localization of diastolic murmurs—Recapitulation of points involved in the localization of systolic and diastolic murmurs—Pathological import of organic endocardial murmurs—Inorganic murmurs—

PAGE

Abnormal modifications of the heart-sounds in cases of valvular lesions—Purring tremor—Diagnostic characters of mitral, aortic, tricuspid, and pulmonic lesions—sudden death in cases of valvular lesions—Treatment of valvular lesions, ... 194

CHAPTER V.

CONGENITAL MISPLACEMENTS, DEFECTS, AND MALFORMATIONS OF THE HEART.

Transpositions and expositions—Ectopia pectoralis cordis—Ectopia cordis ventralis—Ectopia cordis cephalica—Deficiency of the pericardium—Biloculate heart—Heart with three cavities—Deficiency of auricular and ventricular septa—Obliteration of, and obstruction at, the pulmonic artery—Supernumerary septum—Patency of the foramen ovale and the ductus arteriosus—Deficiency and excess of segments of semilunar valves—Union of curtains of the mitral and tricuspid valves—Diagnosis of malformations—Causes of death—Coexistence of tuberculosis—Treatment.

Cyanosis. Definition—Its connection with different malformations—Conclusions respecting the mode of its production—Cyanotic phenomena in various affections exclusive of malformation of the heart—Diversities as respects degree and extent of the cyanotic discoloration—Other variations—Associated symptoms—Diagnosis—Prognosis—Treatment, 260

CHAPTER VI.

CERTAIN AFFECTIONS INCIDENTAL TO DISEASES OF THE HEART.

Coagulation of Fibrin within the Cavities of the Heart.—Clots formed after death and at the close of life—Fibrinous coagula formed during life—Their pathological connections—Their formation in organic affections of the heart—Symptoms denoting their formation—Physical signs and diagnosis—Prognosis—Treatment.

Polypi of the Heart.

Angina Pectoris.—Symptoms characteristic of—Description of paroxysms—Exciting causes—Pathological character and relations—Infrequency of the affection—Influence of age and sex—Gravity and prognosis—Diagnosis—Treatment.

Enlargement of the Thyroid Body and Prominence of the Eyes.—Phenomena descriptive of the prominence of the eyes—Phenomena descriptive of the enlargement of the thyroid body—Other symptomatic phenomena—Pathology—Causation—Diagnosis—Prognosis—Indications for treatment.

Reduplication of the Heart-Sounds.—Different varieties of reduplication and their relative infrequency—Cases of reduplication of both

P

sounds—Cardiac lesions found after death in cases of reduplication—Mechanism of reduplication—Bearing of the facts pertaining to reduplication on the mechanism of the normal heart-sounds—Mode of distinguishing the different varieties of reduplication—Pathological import and diagnostic significance of reduplications—Treatment, . . 2

CHAPTER VII.

INFLAMMATORY AFFECTIONS OF THE HEART—PERICARDITIS.

Acute pericarditis—Anatomical characters—Division into three stages or periods—White spots on the heart—Pathological relations and causation of pericarditis—Connection with acute rheumatism, with albuminuria or Bright's diseases, with endocarditis, &c.—Symptoms of acute pericarditis—Symptoms referable directly to the heart, to the circulation, to the respiratory system, to the digestive system, to the countenance, position, &c., to the nervous system—Notable disorder of the brain and spinal cord in connection with pericarditis—Physical signs of acute pericarditis—Signs furnished by percussion, auscultation, palpation, inspection, and mensuration—Summary of the physical signs of acute pericarditis—Diagnosis of acute pericarditis—Prognosis in acute pericarditis—Treatment of acute pericarditis—Bloodletting, mercurialization, sedatives, revulsives or counter-irritants, opium, stimulants, and eliminatives—Treatment prior to liquid effusion, during the period of liquid effusion, and after absorption of liquid effusion—Treatment when complicated with notable disorder of the nervous system—Subacute and chronic pericarditis, with and without liquid effusion—Symptoms, physical signs, and treatment—Paracentesis of the pericardium—Pneumo-pericardium and pneumo-pericarditis—Pericardial adhesions—Effects upon the heart and circulation—Diagnosis, ... 3

CHAPTER VIII.

INFLAMMATORY AFFECTIONS OF THE HEART. ENDOCARDITIS—MYOCARDITIS.

Endocarditis—Definition—Anatomical characters—Pathological relations and causation—Symptoms—Physical signs—Diagnosis—Prognosis—Treatment. Myocarditis, ... 42

CHAPTER IX.

FUNCTIONAL DISORDER OF THE HEART.

Definition, and the different forms of disorder—Pathological relations and causation of functional disorder—Association with plethora, anæmia, PAGE

various derangements of the nervous system, dyspepsia, gout, &c.—Symptoms of functional disorder—Physical signs furnished by percussion, palpation, and auscultation—Diagnosis of functional disorder—Prognosis—Treatment, ... 456

CHAPTER X.

DISEASES OF THE AORTA.—THORACIC ANEURISMS.

Acute aortitis—Subacute or chronic aortitis—Morbid deposit on the surface of the lining membrane of the aorta—Atheroma—Calcareous deposit—Dilatation of the aorta—Contraction and obliteration of the aorta—Thoracic aneurisms—Definition—Varieties—Anatomical relations, &c. Causes—Terminations—Symptoms—Physical signs—Diagnosis—Treatment, ... 487

DESCRIPTION OF THE PLATE

IN FRONT OF THE TITLE.

FIG. 1 illustrates the relations of the heart to the thoracic parietes. The letters *a*, *b*, *c*, &c., indicate the ribs. The figures 1, 2, 3, &c., mark the intercostal spaces. The vertical line denotes the median line. The right-angled triangle extending over a portion of the surface of the heart represents the "superficial cardiac region" as delineated on the chest with sufficient accuracy for practical purposes. The cross on the fourth rib shows the situation of the nipple. The relations of the ventricles, auricles, apex of the heart, aorta, and pulmonary artery, to the ribs and intercostal spaces, the median line and the nipple, are accurately indicated.

FIG. 2 illustrates the relations of the heart to the pulmonary organs, liver, and stomach. The quadrangular space in which the heart is uncovered by lung is the "superficial cardiac region," represented more accurately than in Fig. 1. The relative situations of the left lobe of the liver, the stomach, and inferior border of the heart, are correctly represented.

DISEASES OF THE HEART.

CHAPTER I.

ENLARGEMENT OF THE HEART.

Definition and varieties of hypertrophy and dilatation—Normal dimensions and weight of the heart—Enlargement by hypertrophy—Concentric hypertrophy—Symptoms and pathological effects of hypertrophy—Physical signs and diagnosis of enlargement and hypertrophy—Situation and anatomical relations of the heart in health—Alterations in degree and extent of dulness on percussion in hypertrophy—Altered situation and extent of the apex-beat, and abnormal force of impulse in hypertrophy, as determined by palpation—Abnormal modifications of the heart-sounds—Diminished extent and degree of the respiratory murmur and vocal resonance within the præcordia in hypertrophy, as determined by auscultation—Results of the clinical study of the heart-sounds in health—Enlargement of the præcordia and abnormal movements in hypertrophy, as determined by inspection—Increased size of the chest, as determined by mensuration—Summary of the physical signs of enlargement of the heart—Summary of the physical signs distinctive of enlargement by hypertrophy—Treatment of hypertrophy—Enlargement by dilatation—Symptoms and pathological effects of dilatation—Physical signs and diagnosis of dilatation—Summary of the physical signs distinctive of enlargement by dilatation—Treatment of dilatation.

ENLARGEMENT OF THE HEART is a term which embraces abnormal increase of this organ, as regards either volume or weight, or, as is commonly the case, increase both in weight and volume. Increase of the volume of the heart, and increase of its weight, are different forms of enlargement, either of which, although they are usually associated, may exist independently of the other. The heart may exceed the limit of health, as regards weight, from an increased thickness of its walls, the normal volume being retained. This is a condition sometimes found after death, although, in the vast majority of the cases in which the weight is augmented, the volume exceeds the healthy limit. On the other hand, the volume of the heart may be abnormally great, the cavities being enlarged, while the thickness of the walls is so far diminished that the normal weight is retained. The latter form of enlargement is also of very rare occurrence, the organ generally increasing in weight when its volume is greater than in health. Abnormal increase of the heart in weight, due to morbid thickness of the muscular walls of the organ, constitutes the morbid condition called *hypertrophy*. Abnormal increase of the heart in volume, due to the morbid size of its cavities, constitutes the morbid condition called *dilatation*. These names, hypertrophy and dilatation, thus denote different forms of enlargement of the heart, which exist sometimes separately, but usually together.

Hypertrophy and dilatation have been subdivided by writers into several varieties, the subdivisions being based on well-marked distinctions. *First.* Hypertrophy exists in some cases without any alteration of the cavities, the latter remaining normal. This has been called *pure* or *simple* hypertrophy. *Second.* The cavities are sometimes found to be diminished in size below the limit of health. This has generally been admitted as a variety of hypertrophy, although its existence, as a morbid condition, is open to doubt. It has been distinguished as *concentric hypertrophy*, or *hypertrophy with contraction*. *Third.* The variety occurring much more frequently than the others is characterized by the coexistence of dilatation to a greater or less extent. This variety has been called *eccentric hypertrophy* or *hypertrophy with dilatation*. Dilatation, on the other hand, differs in different cases, according to the thickness of the walls of the heart. *First.* It exists in some cases, the walls retaining their normal thickness. This has been called *pure* or *simple* dilatation. It is obvious, however, that, in proportion to the dilatation, the heart is hypertrophied, assuming the walls to preserve their normal thickness, inasmuch as the quantity of muscular structure and the weight of the organ, under these circumstances, must be increased. *Second.* In other cases in which the capacity of the cavities is increased, the thickness of the walls is diminished. In this variety, the weight of the heart may not exceed, and may even fall below, that of health. This has been distinguished as *dilatation with attenuated walls*, or *attenuated dilatation*. *Third.* The variety of dilatation which occurs with far greater frequency than either of the other varieties is characterized by increased thickness of the walls, or well-marked hypertrophy, the dilatation, however, being predominant.

These subdivisions, although based on distinctions which are valid, are embarrassing to the student. They are consistent with the different morbid conditions of the heart, as determined by examinations after death; but they are not accompanied by diagnostic criteria, by means of which they may be discriminated at the bedside during life. A simpler arrangement is clinically more available, and suffices for all practical purposes. We may distribute all cases of enlargement of the heart into two groups, 1st. Enlargement by hypertrophy; and 2d. Enlargement by dilatation. These groups will include, respectively, cases in which the hypertrophy or the dilatation is either simple or predominant. In cases of enlargement by hypertrophy, the cavities may, or may not, exceed their normal capacity. Cases in which the cavities are diminished will also fall in this class. If the hypertrophy be neither simple nor *concentric*, it is included in this class whenever it is greater than the coexisting dilatation. The symptoms and signs enable the diagnostician to determine, often with positiveness, the existence of hypertrophy, which may be either simple, or predominant over a coexisting dilatation; but to discriminate between the cases in which the hypertrophy is simple and those in which it predominates over coexisting dilatation, is a problem in diagnosis by no means easily solved. So in cases of enlargement by dilatation, the quantity of muscular structure may, or may not, exceed the limit of health. The diagnostic criteria of predominant dilatation are often sufficiently positive; but it is far less easy to decide whether the dilatation be accompanied with hypertrophy or attenuation. Moreover, as regards prognosis and treatment, after the existence and degree of enlargement are ascertained, it is enough to determine which form of enlargement predominates, namely, either hypertrophy or dilatation. In treating of enlargement of the heart, I shall follow the simple arrangement just indicated.

As a point of departure for the study of enlargement by hypertrophy and by dilatation, the normal volume and weight of the heart are to be considered. The healthy standards in these respects are obtained by measuring and weighing a sufficiently large number of hearts presumed to be free from disease. As regards measurements, the diameters and the thickness of the walls are the points which have reference to the affections to be treated of in this chapter. The dimensions of the orifices and valves will be considered in connection with lesions in this situation. The researches of Bizot and others have shown that the volume of the heart varies according to sex and age. It is somewhat greater in the male than in the female, and it increases slowly, but progressively, from infancy to old age. It is to be observed that diametrical measurements after death are liable to be affected by incidental circumstances, by which they are rendered only approximatively correct. The degree of contraction varies according to the quantity of blood which the cavities contain at the time of death. Observations show that when death occurs from hemorrhage and from diseases attended by rapid loss of fluids, the cavities are much diminished, and the volume proportionately small, whereas, if the cavities be distended with blood, they are dilated, and the volume increased in proportion. In consequence of these variations, the measurements of the entire organ, made by means of careful percussion and auscultation during life, are as reliable, if not more so, than those made in the dead subject. As standards for comparison, with reference to the existence of abnormal enlargement, it is sufficient to take into view the vertical and transverse diameters, the contents of the cavities having been removed; and it suffices to express the normal averages in figures approximating to the exact results obtained by taking the mean of measurements, disregarding fractional amounts, which the student cannot be expected to remember. Adopting, as a basis, the measurements by Bizot and others, it is sufficiently exact to say that the average length of the heart, measured from apex to base on its anterior surface, in the male, between the ages of thirty and fifty, is about four inches, being in the female somewhat less; and that the width, measured at its widest part, in the male, is a small fraction over four inches, being somewhat less in the female.[1]

The general remarks just made with reference to the normal volume of the heart, are also applicable to the thickness of the walls; the thickness is greater, as a rule, in males than in females, and it increases progressively with age. It varies, also, according to the contraction of the heart at the time of death, being dependent on the amount of blood contained within the cavities, and other circumstances. Hence, measurements here, as with respect to the diameters, in a collection of hearts, furnish results which are only approximations to correctness. It is approaching near enough to exactness to say that the thickness of the left ventricle, at its thickest portion, in middle life, is not far from half an inch in the male, and in the female a fraction less. The thickest part of this ventricle is near its centre. The thickness is less near the base, and still less at the apex. The thickness of the right ventricle, at its thickest portion, is a little over one-sixth of an inch, in the male, and in the female somewhat less. The thickest part of this ventricle is near the base, and the thinnest near the apex. The relative thickness of the two ventricles is, thus, in the ratio of 3 to 1. The average thickness of the right auricle is about a twelfth of an inch, and of the left auricle somewhat greater.

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[1] Farther details with regard to measurements of volume are dispensed with as practically not important in this connection. Bizot's extensive and elaborate researches were published in the Mémoires de la Sociéte Médicale d'Observation de Paris, 1836. See also Traité Clinique des Maladies du Cœur, par J. Bouillaud, which contains measurements by himself and strictures on the researches of Bizot.

The average normal dimensions of the heart as a whole, and of different parts of the organ, are important as standards of comparison by which to estimate abnormal changes. Their importance in this respect, however, is less than might, at first view, be imagined. The deviations from these standards, within the limits of health, are to be taken into account. The range of normal variation, as regards the volume of the heart and thickness of its walls, is considerable. An addition of an inch or more to the vertical and transverse diameters may not be abnormal. So, a proportionate amount of increased thickness of the walls of the ventricles may be within healthy limits. To determine the line of demarcation between normal and abnormal deviations is more difficult than to ascertain averages. It is not easy to fix a maximum and a minimum, beyond which the condition is always morbid. And even were the boundaries definitely fixed, it might still be a matter of doubt in some individual cases in which these were not exceeded, whether the condition was not abnormal. Enlargement of the heart sufficient to be of pathological importance is generally so well marked that its existence does not admit of doubt. Practically, therefore, the inability to define rigorously the confines of morbid anatomy, does not lead to serious inconvenience. These statements are applicable, not only to the dimensions of the heart already considered, but, equally, to the capacity of its cavities and to its weight.

The cavities of the heart are not readily measured. Their capacity varies, irrespective of intrinsic normal differences, according to the quantity of blood which they contain, and the condition of the muscular walls at the time of death. They are also affected by post-mortem changes. The two ventricles and auricles, in health, present a variable amount of disparity in capacity. The right ventricular and auricular cavity are larger than the left, namely, in the proportion of one-tenth to one-third. Dilatation, when it exists to an extent to constitute a lesion of importance, and as it is met with in autopsies of subjects dead with cardiac disease, is sufficiently well marked to be recognized, and its degree may be determined by the eye with sufficient accuracy for all practical purposes.

The average weight of the heart, as determined by weighing a large number presumed to be free from disease, and taking the mean, is not easily fixed with precision, because the results in different hands differ considerably, a fact which goes to show that the variations within the limits of health are considerable. For the reasons, however, which were stated with respect to the average size of the organ, mathematical exactness in giving the average weight is not practically important. The range of normal variation is more important to be considered. It is sufficient to say that the average weight is between eight and ten ounces. And it is to be borne in mind that if it be found to exceed this average, or fall below it, by one or two ounces, it is by no means to be inferred that the condition is abnormal. The medium weight in the female is somewhat less than in the male. The weight, as well as the dimensions of the heart, increases progressively up to an advanced period of life.

ENLARGEMENT BY HYPERTROPHY.

Under this head, it is to be borne in mind, I include not only enlargement due exclusively to increased thickness of the muscular walls, but enlargement by hypertrophy with dilatation, provided the former predominate over the latter. In examining the heart, after laying open the cavities and removing their contents, the predominance of either hypertrophy or dilatation is generally obvious to the eye. The two forms of enlargement are combined, in different cases, in every degree of relative proportion. In determining, then, whether the enlargement be by hypertrophy or by dilatation, the question is, which contributes most to the morbid size, increase of the structure, or increased capacity of the cavities. Instances, however, occur in which these two forms of enlargement are about evenly balanced. On measuring and weighing the heart, the excess of weight is greater than the abnormal dimensions in proportion as hypertrophy preponderates. The walls are more solid and resisting. The ventricles retain their rounded form when the heart is placed on its posterior surface, not being flattened by the collapse of the ventricular walls. If the increased thickness of the walls of the ventricles be due purely to hypertrophy, they present externally, and on section, the appearances of healthy muscular structure, and the microscope shows the characters of the normal tissue; the hypertrophy causing abnormal volume and weight is due to increase of the muscular substance. The heart may be more or less enlarged by an accumulation of fat upon the surface and between the muscular fibres, or by the presence of different morbid products in these situations. Under these circumstances, notwithstanding the abnormal volume and weight, the muscular substance may be diminished; that is, instead of hypertrophy, there is atrophy of the heart.

The several portions of the heart may collectively participate in the enlargement, or it may be confined to one or more of the anatomical divisions without extending to the whole organ. In the majority of cases all portions are involved, but they are rarely affected equally; the enlargement is more marked in some divisions than in others. The different portions may not present the same form of enlargement. Hypertrophy may predominate in one part and dilatation in another. If the enlargement be limited to, or be seated chiefly in, the left ventricle, the vertical is more increased than the transverse diameter; the heart is elongated, and the conoidal form may be more marked than in health. If the enlargement be great, the right appears to be merely an appendix of the left ventricle. The apex is lowered, and is more or less removed to the left of its normal situation. On the other hand, if the enlargement be limited to, or be seated chiefly in, the right ventricle, the width more than the length is increased; the conoidal form is less marked than in health, and the apex, formed in part or entirely by the right ventricle, is blunt instead of pointed. The apex extends lower than in health and more in a direction toward the epigastrium than when the enlargement is seated in the left ventricle. If both ventricles be considerably enlarged the organ has a globular form. The papillary muscles are not infrequently more or less increased in size when the ventricular walls are thickened. The degree of hypertrophy varies greatly in different cases. The thickness of the left ventricle may be increased to an inch, an inch and a half, and even two inches. The walls of the other compartments may, in like manner, be doubled, tripled, and quadrupled. The vertical and transverse dimensions may be five or six inches, or more. The weight may exceed two, three, four, and even more than five times the normal average.

Is enlargement by hypertrophy due to an increase in size of the muscular fibres, or does it involve an abnormal multiplication of the fibres? If the term hypertrophy be applied exclusively to morbid growth, it implies that the enlargement is due to the former, that is, to an increase in size of the muscular fibres. The term is thus restricted by Virchow, and others; the multiplication of fibres, on the other hand, is called hyperplasia, or hypergenesis. Measurements in normal hearts, and in hearts more or less hypertrophied, show an increase of size of the muscular fibres, their diameter in the latter sometimes being four times greater than in the former.[1] The heart may, therefore, be enlarged so that the volume will be four times greater than in health by hypertrophy, in the restricted sense of the term. This, however, will probably not account for the increase of the muscular substance in all cases, and, if not, the multiplication of fibres must be admitted. Enlargement of the heart by hypertrophy, therefore, may be due wholly to hypernutrition, or increased growth of the muscular fibres, and hyperplasia may be superadded.

What causes the pathological processes, namely, hypernutrition and hyperplasia, which increase the quantity of the muscular substance of the heart? Generally, if not invariably, enlargement by hypertrophy is the result of prolonged abnormal force of the heart's action. It is difficult to account for this form of enlargement, except as caused by augmented muscular power continued for a long period; and generally there are present obvious causes which account, in this way, for the enlargement. The mechanism is the same as in the familiar examples of certain voluntary muscles becoming disproportionately developed when inordinately exercised. The muscles of the arms of the blacksmith are strikingly in contrast with the muscles of the lower limbs; and the reverse is true of pedestrians and dancers. Involuntary muscles, aside from the heart, also present examples. For instance, the muscular structure of the urinary bladder may become enormously hypertrophied, when the power of contraction of this organ has been for a long time increased in consequence of obstruction to the expulsion of the urine. Clinical observation shows that in most cases of enlargement of the heart by hypertrophy, there are prior morbid conditions which stand to it in a causative relation. The practical bearing of this pathological view of hypertrophy is vastly important. It follows, that enlargement of the heart by hypertrophy, as a rule, is compensatory, or, in other words, a conservative provision to meet the difficulties incident to the morbid conditions upon which the hypertrophy depends. This truth cannot be too strongly impressed.

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[1] Raynaud, vide Nouveau Dictionnaire de Médecine et de Chirurgie. Paris, 1868.

In the great majority of cases, enlargement by hypertrophy is consecutive to, and dependent upon, morbid conditions within the heart, namely, on valvular lesions. These give rise to hypertrophy when they involve over-repletion of the cavities in consequence either of obstruction to the free passage of the blood through the orifices, or of regurgitation due to valvular insufficiency. The organ being unduly distended and stimulated by the accumulation of blood, its action becomes abnormally forcible; the causes of accumulation being permanent and often progressively increasing, the increased power of action continues and augments, and hypertrophy is the result. The hypertrophy commences in that portion of the heart which is primarily affected, but the several portions sustain to each other, in their anatomical structure and functions, relations so close and reciprocal, that causes which at first are limited to one portion, affect ultimately the whole organ. The enlargement, however, preponderates in the portion which is first affected. Directing attention with some detail to the mode in which valvular lesions give rise to enlargement, we shall be led to consider the development of the affection in the different anatomical divisions of the heart, respectively, taking them up in the order of their greater relative liability to become hypertrophied. Of the several portions, the left ventricle is oftenest enlarged; next in liability to enlargement is the left auricle; next, the right ventricle, and last, the right auricle.

The valvular lesions which especially lead to hypertrophy of the left ventricle, are seated at the aortic orifice. Lesions in this situation may involve, as will be seen hereafter, either contraction and consequently obstruction, or incompetence of the valves and consequent regurgitation of blood from the aorta into the ventricular cavity. Contraction and valvular insufficiency are not infrequently combined, causing, at the same time, both obstruction and regurgitation. Either of these immediate effects of aortic lesions occasions over-repletion of the left ventricle; hence, undue distension and stimulation, followed by undue force of the ventricular contractions, and, sooner or later, hypertrophy results, often accompanied with more or less dilatation. The enlargement, for a time, is limited to the left ventricle. Eventually the other portions are likely to become enlarged. The right ventricle is affected because each of the two ventricles participates in the action of the other. The two not only contract synchronously, but are in part composed of muscular fibres common to both. Hence, causes which increase the force of the contractions of the one, exert, to a greater or less extent, a similar effect on the contractions of the other. Clinical observation shows that, with enlargement of one ventricle, the other rarely retains its normal size. This is a mode by which the enlargement is extended, applicable only to the ventricles. Another mode is more effective than this. The accumulation of blood within the cavity of the left ventricle offers an obstacle to the free transmission from the left auricle. The blood in passing from the auricle to the ventricle meets with an obstruction in the already repleted ventricle. Over-accumulation within the left auricle ensues; hence occurs, after a time, enlargement of the auricle. This enlargement involves generally more or less thickening of the walls, but dilatation here uniformly predominates over hypertrophy. Enlargement by hypertrophy, in fact, pertains exclusively to the ventricles. Persisting repletion of the left auricle offers an obstacle to the free transmission of blood from the lungs; hence arises congestion of the pulmonary vessels proportionate to the over-accumulation of blood within the auricle. Congestion of the pulmonary vessels offers an obstacle to the current propelled by the right ventricle into the pulmonary artery; hence, undue distension and excitement of the right ventricle, leading ultimately to enlargement of this portion of the heart. Over-accumulation within the right ventricle offers an obstacle to the passage of the blood from the right auricle into that cavity; hence result, at length, dilatation and thickening of the walls of the right auricle. Over-accumulation in this auricle induces congestion of the systemic and portal veins. This congestion offers an obstacle to the free passage of blood through the arteries of the larger circuit. Finally, this latter obstacle reacts on the left ventricle, and adds to the accumulation in that portion, where commenced the several links in the chain of sequences tending to the enlargement, successively, of all the other portions of the heart. And while the whole organ thus becomes implicated, the causes affecting primarily the left ventricle are more and more operative, giving preponderance to the enlargement of the latter. The enlargement of the left ventricle, and, sequentially, of the remainder of the organ, will be, *cœteris paribus*, proportionate to the duration and degree of the aortic contraction or insufficiency, or of both combined. The amount of over-accumulation in the several cavities, and the enlargement of the different portions of the heart, the latter due to the obstruction caused by the over-accumulation successively in the several cavities, will, of course, be greater in proportion as dilatation coexists with hypertrophy.

Obstruction seated in the aorta, either near to or at some distance from the heart, such as is incident to aortic aneurism, may lead to hypertrophy of the left ventricle primarily, and, subsequently, of the other portions. Atheromatous and calcareous disease of the aorta may also have the same effect, the diminished elasticity of the walls of this vessel constituting an obstacle to the circulation, and thus leading to increased power of the contraction of the left ventricle. Obstruction in the aorta from aneurism and diminished elasticity of the walls are often associated with lesions at the aortic orifice, involving either contraction, or regurgitation, or both.

Enlargement commences in the left auricle in connection with lesions affecting the mitral orifice and valves, and involving either contraction or insufficiency, or both these immediate effects. In enlargement of the auricles, however, as just stated, dilatation predominates over hypertrophy. Mitral contraction and regurgitation lead to accumulation in the left auricle, the passage of the blood from the auricle to the ventricle being impeded by obstructive lesions, and a retrograde current from the ventricle to the auricle being incident to lesions which render the valves incompetent. Next follow pulmonary congestion, and consequent upon this, enlargement of the right ventricle. So far as the ventricles are concerned in connection with mitral lesions, the right ventricle is first enlarged, and its enlargement preponderates over that of the left ventricle, unless, as frequently occurs, aortic lesions also exist. The enlargement of the right, however, leads ultimately to that of the left, ventricle, partly from the muscular fibres common to both ventricles, and in part from the ultimate effect on the left ventricle of obstructive accumulation successively in the right auricle and the systemic veins.

Contraction and valvular insufficiency at the orifice of the pulmonary artery, occasion, primarily, enlargement of the right ventricle, precisely as aortic lesions induce, first, enlargement of the left ventricle. Lesions at the pulmonic orifice after birth, however, are so rarely met with that, practically, their occasional occurrence may almost be disregarded in diagnosis. In fœtal life, contraction at this orifice is not very infrequent. It is the point of departure for many of the congenital malformations of the heart. In these cases the right ventricle often becomes enormously hypertrophied.

Lesions at the tricuspid orifice being extremely infrequent, enlargement of the right auricle rarely occurs, except consecutively to an affection of the right ventricle. Over-accumulation in this ventricle involves obstruction and accumulation within the auricle with which it communicates, together with the ulterior consequences already stated. The remote and incidental effects of obstruction to the circulation, except as regards the size of the heart, will be considered, in connection with valvular lesions, in another chapter.

Enlargement of the heart, not associated with valvular lesions, may be due to obstruction at a distance from the centre of the circulation. Obstruction to the pulmonary circulation incident especially to emphysema of the lungs, and occasionally to chronic pleurisy, collapse, and dilated bronchi, leads to enlargement. In these cases, the point of departure is the right ventricle, and the enlargement of this portion preponderates over that of the other portion of the heart. In some cases of emphysema, this affection having existed for a long period, the right ventricle and auricle become greatly enlarged both by hypertrophy and dilatation, giving rise to general dropsy, the left ventricle and auricle becoming but little or not at all enlarged. On the other hand, enlargement, either limited to, or predominating in, the left side of the heart, occurs so often as a concomitant of chronic Bright's diseases, and so infrequently, irrespective of valvular and aortic lesions, except in association with these diseases, that it is fair to infer the existence of a pathological connection. Clinical facts warrant the conclusion that, in these cases, the enlargement of the heart is consecutive to, and dependent upon, the renal diseases. Probably, as conjectured by Bright, these diseases give rise to enlargement of the left side of the heart by occasioning changes in the blood, which impede its free passage through the systemic capillaries, the increased arterial tension occasioning augmented power of contraction in the left ventricle. The theory of Traube, that the impaired functional activity of the kidneys diminishes the flow of blood in the renal arteries and induces an accumulation of water in the blood sufficiently to give rise to enlargement by increasing the arterial tension, is hardly tenable. Hypertrophy of the heart is not found to follow the interruption of the flow of blood through arterial vessels larger than the renal, as when large arteries are ligated, or limbs are amputated; and, as regards the accumulation of water in the blood, the heart becomes hypertrophied in cases of renal disease in which there is no deficiency of water in the urine. The changes which the arteries undergo in the latter part of life, by which their elasticity is impaired and their calibre diminished, are, with much reason, supposed to stand in a causative relation to enlargement of the heart in some cases. These changes, in a measure at least, account for the progressively increasing size of the heart, which marks the progress from middle life to old age.

The researches of Larcher, Ducrest, and others, show that a certain amount of hypertrophy, limited to the left ventricle, is incident to pregnancy.[1] It would seem that the hypertrophy, under these circumstances, is to be regarded as normal, and that it disappears after confinement. The changes which occur in the muscular structure of the uterus, in connection with gestation, are thus represented on a small scale in the heart. The increase in weight of the heart in pregnancy, it is estimated, may amount to one-fifth of the previous weight of the organ. Doubtless this temporary hypertrophy is compensatory or conservative, as it is when it occurs in other connections. Here too, as in other connections, it is doubtless the result of augmented power of the contraction of the left ventricle, occasioned chiefly, or in part, by the increased area of the arterial system, and perhaps partly by increased arterial tension due to the pressure of the gravid uterus on the iliac arteries.

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[1] Vide Archives de Médecine. Paris, May, 1859, p. 291.

It was formerly supposed that prolonged functional disorder of the heart frequently eventuated in the development of hypertrophy. This supposition does not derive much support from clinical experience. At first view, the statement just made, may appear inconsistent with the fact that the abnormal growth of the muscular walls of the heart is the result of abnormal muscular action of the organ. The inconsistency disappears when it is considered that functional palpitation, even when intense, does not involve that increase of power or strength of muscular action which is incident to the over-accumulation of blood from an impediment to the circulation. Moreover, the increased action from nervous excitation is rarely as constant and persisting as that due to valvular or other lesions which occasion obstruction. In the latter case, hypertrophy is the result of increased force of action, beginning imperceptibly and progressively increasing for many months, and even years. Excessive action of the heart, however, without augmented power, if continued for a sufficiently long period, may lead to enlargement. An illustration of this fact is afforded by a case of persistent frequency of the heart's action accompanied by prominence of the eyes and enlargement of the thyroid body, in the affection sometimes called "Graves' disease." The case first came under my observation in 1861. At this time there was no enlargement of the heart. In 1862 the case again came under my observation, and there were no signs of enlargement. A year afterward the signs of slight enlargement were noted. Five years afterward, that is, in 1868, the case again came under my observation, and now the signs denoted considerable enlargement. The signs denoting considerable enlargement were verified a year later, namely in 1869. At each examination the action of the heart was rapid, the pulse never being below 120 per minute. There have never been any signs denoting valvular lesions in this case; nor have there been any obvious causes of the enlargement, which has thus taken place under my observation, excepting the persistent frequency of the heart's action. Dr. J. M. Da Costa has reported cases in which hypertrophy, supposed to be limited to the left ventricle, was developed in soldiers after long and heavy marching. This able clinical observer and writer is also of the opinion that persistent functional disorder, especially after convalescence from fevers, was, in some cases, the cause of hypertrophy.[1]

Enlargement by hypertrophy, as already stated, is almost always a secondary affection. In the great majority of cases, it is consecutive to valvular or aortic lesions. It is also an effect of certain chronic pulmonary diseases, more especially emphysema of the lungs. It occurs in certain cases of Bright's diseases. It is a physiological event in pregnancy. It may be produced, but the examples are very rare, by long persisting functional disorder. Its occurrence, when it is not evidently a secondary affection, is so infrequent that there is room for doubting whether it ever be a truly idiopathic affection. In other words, considering the fact that it is almost always secondary, to suppose it to be so when previous affections are not apparent, is perhaps more reasonable than to suppose that it is primary in these cases. However this may be, hypertrophy sometimes occurs when it cannot be referred to any prior affection, either within the heart or elsewhere. Instances are on record in which the heart has been found to be enormously hypertrophied without valvular lesions or any morbid conditions to account for the enlargement. The following is a striking instance within my cognizance: I. S., aged 23, residing in Brooklyn, breakfasted with his family, and shortly after breakfast left for New York, apparently in good health, to enter for the first time upon duties connected with a new mercantile arrangement. He had not proceeded far when he was noticed to stagger and fall heavily forward on the sidewalk. He was almost immediately taken to a police station-house near at hand, but he expired before reaching it. A post-mortem examination was made by Prof. William Gilfillan, eight hours after death. The face was swollen and livid. Blood oozed freely from the nostrils and from both ears.

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[1] Vide article in "Sanitary Memoir of the War of the Rebellion. Collected and published by the United States Sanitary Commission." 1867, p. 373.

The incision through the scalp, and the removal of the calvarium occasioned a profuse flow of blood. The bloodvessels of the superficies of the brain were greatly engorged. There was no other morbid appearance within the skull. The ventricles did not contain liquid. The substance of the brain was firm and healthy. The pericardium contained but little liquid. The heart presented considerable fulness of the superficial vessels. The length of the organ was 6¾ inches, and the width 5 inches. The left ventricle at its thickest point measured 1⅝ inches. The thickness of the right ventricle was ⅜ inch. The aortic valves were competent, as shown by the water test. The mitral orifice and valves were normal. The pulmonic and the tricuspid orifice presented no morbid appearance. The left cavities were free from blood. The right auricle was much distended. The heart weighed 15½ ounces. The cavities were not dilated. The left ventricular cavity, indeed, appeared to be diminished in size. The lungs were healthy. The kidneys were of normal size and presented no appearance of disease. The muscular structure of the heart was normal.

Dr. William H. Dudley, to whom I am indebted for this history, had known the person from boyhood. He had never had any acute disease, and he appeared to be always in excellent health. He was accustomed to take very active exercise without any inconvenience; and he complained of no ailments pointing to an affection of the heart.

The heart, which was kindly placed at my disposal, was presented by me at a meeting of the New York Pathological Society, June 11th, 1862.

Perhaps the most rational explanation which can be given of the hypertrophy in cases like this is that, congenitally, the size of the heart is disproportionate to the capacity of the vascular system.

The account which has been given of the manner in which the several portions of the heart become enlarged, is applicable to both forms of enlargement, viz., hypertrophy and dilatation. The latter will be considered in a subsequent section of this chapter, devoted to the subject of "enlargement by dilatation."

In the cases of enlargement by hypertrophy, in which there is more or less coexisting dilatation, the dilatation, according to the views of some writers, precedes the hypertrophy. It is more reasonable to suppose the reverse of this, namely, that the dilatation is consecutive to the hypertrophy. The first effect of over-distension and stimulation from an undue accumulation of blood is increased growth of the muscular walls. In the healthy, vigorous action of the heart, the ventricles probably contract so that the endocardial surfaces come into apposition, and the contents of the cavities are completely expelled.[1] Over-repletion of the cavities excites a more forcible ventricular action which for a time enables the ventricles to expel their contents. Meanwhile, hypernutrition follows, and hypertrophy is produced. The increased muscular growth for a certain period protects against the occurrence of dilatation. At length, the hypertrophy reaches a point beyond which it cannot advance; for the muscles of the heart, like other muscles, cannot increase indefinitely. There is a limit to hypertrophic enlargement, and this limit varies in different persons, just as the voluntary muscles in different persons attain, by the same efforts, to different degrees of development. The causes, however, persist, and perhaps become more and more operative after the utmost degree of hypertrophy which is possible has taken place. These causes then can produce only dilatation, and from this period the progressive enlargement is due to augmentation of the cavities. This view is not only rational, but sustained by facts derived from clinical experience. Observation shows that, as a rule, in proportion to the duration of organic affections of the heart inducing enlargement, dilatation exceeds, relatively, hypertrophy; and, in the great majority of the cases in which death occurs, not from affections incidental to heart disease, but as a termination of the latter, dilatation predominates over hypertrophy. According to this view, hypertrophy becomes an important conservative provision, first, against over-accumulation of blood, and second, against the more serious form of enlargement, viz., dilatation.

Hypertrophy with diminution of the size of the cavities claims a few words. Under the name "concentric hypertrophy," this was formerly recognized as a variety of hypertrophy occurring not very infrequently. The investigations of Cruveilhier and others within the past few years have led some pathologists to reject it entirely as a morbid condition, and it is generally conceded that, if it ever occur, the instances are extremely rare. The ventricular cavities, in connection with increased thickness of the walls, are sometimes observed after death to be considerably diminished. This fact is not doubted; but it is supposed that both the diminished cavities and the thickened walls in such cases are due to an unusual degree of tonic contraction of the muscular fibres persisting after death. Cruveilhier found this appearance in the bodies of persons who had suffered death by decapitation. It has been observed after death from hemorrhage, and from diseases accompanied with much loss of fluids. In some instances, the contracted size of the cavities may be made to disappear by mechanical dilatation with the fingers, and it may disappear spontaneously some time after death, especially if the heart be macerated in water. The coexistence of contracted cavities and morbid thickness of the walls is deemed inconsistent with the conditions giving rise to hypertrophy, and the mechanism of its production. The tendency of these conditions, in most cases, is, undoubtedly, to dilatation. Yet, it is conceivable that causes which have induced hypertrophy without dilatation may cease, and that afterward the tendency of the hypertrophy is to lessen the ventricular cavities. This is the more intelligible when it is considered that, according to the view which has been presented in the development of hypertrophy and dilatation, the former in point of time takes precedence. Hypertrophy of the left ventricle, with contraction of the cavity, may be accounted for in cases in which there exists either mitral contraction or regurgitation. This ventricle, under these circumstances, may become hypertrophied in the manner already considered, while, owing to contraction at the mitral orifice, or regurgitation, the accumulation within its cavity, instead of being sufficient to occasion distension, for a time, at least, is less than normal, and, therefore, the tendency of the hypertrophy, while this state of things continues, may be to contraction rather than dilatation. Without discussing the subject, which does not possess much practical importance, the possibility of concentric hypertrophy must be admitted, while it is probable that, in the majority of the cases formerly so considered, the appearances after death do not fairly represent either the capacity of the cavities or the thickness of the walls during life. It is to be borne in mind that, in the cases in which unusual tonic contraction of the ventricles is suspected, the thickness of the walls may not be adequate evidence of the existence of hypertrophy. The weight of the heart is the test in such cases. If the weight exceed the limit of health, without reference to the size of the cavities or thickness of the walls, it is to be concluded that hypertrophy exists.

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[1] That the inner surfaces of the ventricles come into contact, and with considerable force, was shown by an appearance presented in a heart contained in my collection. A rough, projecting, calcareous deposit existed on the anterior curtain of the mitral valve. Directly opposite, on the septum, over a space corresponding in size, as well as situation, to this deposit, the endocardium had become thickened and opaque, evidently due to the forcible pressure of the rough, calcareous mass. The ventricle was hypertrophied and dilated.

SYMPTOMS AND PATHOLOGICAL EFFECTS OF HYPERTROPHY.

The symptoms of hypertrophy, in the cases which come under the cognizance of the physician, are generally intermingled with those of concomitant cardiac or other affections of which the hypertrophy is an effect. Cases of hypertrophy not associated with, and dependent upon, other affections, are so rare that its clinical history cannot be said to have been established by observation. The symptomatic phenomena which are described as distinctive of it are determined inferentially rather than by facts observed in well-authenticated cases. Rationally considered, it is clear that the symptoms would be those indicative of abnormal power of the heart's action. Undue determination of blood to the head might be expected to occasion certain phenomena, such as cephalalgia, flushing of the face, throbbing, epistaxis, vertigo, &c. These symptoms have relation especially to hypertrophy affecting the left ventricle. Assuming the absence of aortic and of mitral lesions involving obstruction or regurgitation, the pulse would represent by its force, fulness, and incompressibility, the power of the ventricular systole. Dyspnœa, when, from any cause, the action of the heart is increased, as, for example, after exercise, would denote that the hypertrophy affected the right ventricle. Of the powerful action of the heart the patient would be conscious when his attention was directed to it, and it would be apparent from the movements of parts of the body and the dress. The digestive and assimilative functions would not be expected to offer any marked symptoms of disorder. The muscular strength would not be diminished, nutrition would not be impaired, nor the functions of secretion and excretion interrupted. This is a synopsis of a hypothetical case of idiopathic hypertrophy accompanied with little or no dilatawas formerly recognized as a variety of hypertrophy occurring not very infrequently. The investigations of Cruveilhier and others within the past few years have led some pathologists to reject it entirely as a morbid condition, and it is generally conceded that, if it ever occur, the instances are extremely rare. The ventricular cavities, in connection with increased thickness of the walls, are sometimes observed after death to be considerably diminished. This fact is not doubted; but it is supposed that both the diminished cavities and the thickened walls in such cases are due to an unusual degree of tonic contraction of the muscular fibres persisting after death. Cruveilhier found this appearance in the bodies of persons who had suffered death by decapitation. It has been observed after death from hemorrhage, and from diseases accompanied with much loss of fluids. In some instances, the contracted size of the cavities may be made to disappear by mechanical dilatation with the fingers, and it may disappear spontaneously some time after death, especially if the heart be macerated in water. The coexistence of contracted cavities and morbid thickness of the walls is deemed inconsistent with the conditions giving rise to hypertrophy, and the mechanism of its production. The tendency of these conditions, in most cases, is, undoubtedly, to dilatation. Yet, it is conceivable that causes which have induced hypertrophy without dilatation may cease, and that afterward the tendency of the hypertrophy is to lessen the ventricular cavities. This is the more intelligible when it is considered that, according to the view which has been presented in the development of hypertrophy and dilatation, the former in point of time takes precedence. Hypertrophy of the left ventricle, with contraction of the cavity, may be accounted for in cases in which there exists either mitral contraction or regurgitation. This ventricle, under these circumstances, may become hypertrophied in the manner already considered, while, owing to contraction at the mitral orifice, or regurgitation, the accumulation within its cavity, instead of being sufficient to occasion distension, for a time, at least, is less than normal, and, therefore, the tendency of the hypertrophy, while this state of things continues, may be to contraction rather than dilatation. Without discussing the subject, which does not possess much practical importance, the possibility of concentric hypertrophy must be admitted, while it is probable that, in the majority of the cases formerly so considered, the appearances after death do not fairly represent either the capacity of the cavities or the thickness of the walls during life. It is to be borne in mind that, in the cases in which unusual tonic contraction of the ventricles is suspected, the thickness of the walls may not be adequate evidence of the existence of hypertrophy. The weight of the heart is the test in such cases. If the weight exceed the limit of health, without reference to the size of the cavities or thickness of the walls, it is to be concluded that hypertrophy exists.

SYMPTOMS AND PATHOLOGICAL EFFECTS OF HYPERTROPHY.

The symptoms of hypertrophy, in the cases which come under the cognizance of the physician, are generally intermingled with those of concomitant cardiac or other affections of which the hypertrophy is an effect. Cases of hypertrophy not associated with, and dependent upon, other affections, are so rare that its clinical history cannot be said to have been established by observation. The symptomatic phenomena which are described as distinctive of it are determined inferentially rather than by facts observed in well-authenticated cases. Rationally considered, it is clear that the symptoms would be those indicative of abnormal power of the heart's action. Undue determination of blood to the head might be expected to occasion certain phenomena, such as cephalalgia, flushing of the face, throbbing, epistaxis, vertigo, &c. These symptoms have relation especially to hypertrophy affecting the left ventricle. Assuming the absence of aortic and of mitral lesions involving obstruction or regurgitation, the pulse would represent by its force, fulness, and incompressibility, the power of the ventricular systole. Dyspnœa, when, from any cause, the action of the heart is increased, as, for example, after exercise, would denote that the hypertrophy affected the right ventricle. Of the powerful action of the heart the patient would be conscious when his attention was directed to it, and it would be apparent from the movements of parts of the body and the dress. The digestive and assimilative functions would not be expected to offer any marked symptoms of disorder. The muscular strength would not be diminished, nutrition would not be impaired, nor the functions of secretion and excretion interrupted. This is a synopsis of a hypothetical case of idiopathic hypertrophy accompanied with little or no dilatation. The group of symptoms is not highly distinctive; the affection would be likely to be overlooked, and, if the hypertrophy were but moderate in degree, the immediate inconveniences would probably not be sufficient to lead the patient to seek for medical advice. Of this fact, the case, an account of which has been given (see page 32), affords an illustration.

Associated with valvular lesions, emphysema, aneurism, and Bright's diseases, as antecedent and causative affections, the symptoms distinctly referable to hypertrophy are few. The cerebral symptoms are, in general, attributable to obstructed circulation rather than to an abnormal power of the heart. The same remark applies to dyspnœa and other pulmonary symptoms. Valvular obstruction and regurgitation modify, in a marked degree, the characters of the pulse. In short, that which chiefly possesses significance is the evidence afforded by observation and the consciousness of the patient that the heart habitually acts with undue strength. To this the mind of the patient becomes accustomed, and he often appears unconscious of it, even when it is very marked on a physical examination of the præcordia. This evidence of hypertrophy lessens in proportion as it is accompanied by dilatation, and finally disappears when the latter predominates.

The pathological effects of hypertrophy are to be disconnected from those of concomitant affections and accompanying dilatation. Thus isolated, it is not easy to impute to it any special or very important pathological effects. It has been supposed that hypertrophy of the left ventricle sometimes leads to cerebral apoplexy, due to extravasation of blood or congestion, in consequence of the force with which the current of blood is propelled into the vessels of the brain. That cerebral apoplexy may be an effect of disease of the heart is not to be denied, but the cardiac affections which more especially tend to produce it, are those involving obstruction to the return of blood from the head. Moreover, in the great majority of cases, hypertrophy of the left ventricle is associated with either aortic obstruction or regurgitation, or both, and under these circumstances, the strain upon the coats of the cerebral arteries is not commensurate with the force of the ventricular contractions. Statistical researches show that the occurrence of apoplexy in connection with affections of the heart is not proportionate to the degree of hypertrophy.[1] That hypertrophy of the left ventricle, when it is not associated with obstructive or regurgitant lesions, may give rise to congestive apoplexy, is exemplified by the case just referred to (page 32). In this case, there seemed to be no other explanation of the apoplectic seizure producing sudden death, and the hypertrophy in this case was apparently idiopathic. Among my recorded cases is a case of apoplexy with large cerebral hemorrhage, in which there was considerable hypertrophy of the left ventricle, and no valvular lesion. In this case the health was apparently good at the time of the apoplectic seizure. But the patient was sixty years of age, and the arteries of the brain were calcareous; so that, if the hypertrophy of the heart had any agency in producing the cerebral hemorrhage, it was probably an auxiliary cause only, since extravasation of blood into the substance of the brain is not infrequently an effect of calcareous disease of the cerebral vessels without the coexistence of hypertrophy of the heart.

Hypertrophy of the right ventricle has been supposed to give rise to bronchorrhagia and pneumorrhagia. But clinical observation shows that these effects very rarely, if ever, take place, except when (as is often the case) with hypertrophy of the right ventricle is conjoined obstruction at the mitral orifice. The latter involves an impediment to the pulmonary circulation more likely to give rise to hemorrhage than the force with which the blood is propelled by the hypertrophied ventricle. Dropsical effusion into the areolar tissue and serous cavities (general dropsy) is a common effect of organic disease of the heart. It is not, however, an effect attributable to hypertrophy. Hypertrophy existing alone is incapable of producing it. When it occurs in connection with enlargement, it is due either to obstruction from valvular lesions together with dilatation, or to dilatation of the right side of the heart without valvular lesions.

Hypertrophy of the heart, not consecutive to either valvular or aortic lesions, and not an effect of either emphysema or any other pulmonary disease, constitutes always presumptive evidence of the existence of renal disease. It is to be borne in mind that, when secondary to valvular and aortic lesions, or to pulmonary disease, the hypertrophy is to be regarded in the light of a conservative provision, the heart acquiring increased power to carry on the circulation notwithstanding the impediment occasioned by these prior and causative affections. This is probably not less true when the hypertrophy occurs in connection with Bright's diseases, whatever be the mode in which these diseases give rise to it. Hence, in Bright's diseases, as in the other pathological connections, so far from the hypertrophy being productive of symptoms or pathological effects referable to the circulation, it affords protection against them. It is, under these circumstances, an advantage, not an evil. Whenever, in Bright's diseases, enlargement of the heart contributes to general dropsy, and occasions dyspnœa, it is not on account of the hypertrophy, but because either dilatation has become the predominant form of enlargement, or the muscular structure is the seat of degenerative changes which have impaired the power of the heart's action.

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[1] See Walshe on Diseases of the Lungs and Heart, second edition, for an analysis of cases collected from different authors, the results appearing to show that hypertrophy has little or no effect in determining the occurrence of apoplexy.

PHYSICAL SIGNS OF ENLARGEMENT OF THE HEART. SIGNS DISTINCTIVE OF ENLARGEMENT BY HYPERTROPHY.

The physical signs of enlargement of the heart are common to both forms, viz., hypertrophy and dilatation. After having considered these signs in the present connection, it will only be necessary to refer to them briefly in treating afterward of dilatation. Incidental to their consideration will be noticed the signs distinctive of enlargement by hypertrophy. The different methods of physical exploration contribute evidence of enlargement and of hypertrophy. Enumerating them in the order of their relative importance, the methods available in the diagnosis are, percussion, palpation, auscultation, inspection, and mensuration. The signs obtained by these different methods may be conveniently classified as follows: 1. Extended and increased dulness in the præcordia, as determined by percussion. 2. Altered situation and extent of the apex-beat; impulses elsewhere than over the apex of the heart, and abnormal force of impulse, as determined by palpation. 3. Abnormal modifications of the heart-sounds; diminished extent and degree of the respiratory murmur and vocal resonance within the præcordia, as determined by auscultation. 4. Enlargement of the præcordia and abnormal movements, as determined by inspection. 5. Increased size of the chest, as determined by mensuration.

1. *Extended and increased dulness in the præcordia, as determined by percussion.*

A practical knowledge of the extent and degree of the præcordial dulness in health, is an essential preparation for the study of the signs of disease furnished by percussion. With reference to percussion in health, the position of the heart and its anatomical relations to the lungs and the thoracic walls are to be considered.[1]

The heart is situated between the cartilages of the third and sixth ribs. The upper extremity, or base, is defined with sufficient precision by the upper margin of the third rib. The point or apex generally extends to the fifth intercostal space, near the junction of the rib to its cartilage. The organ is situated obliquely within the chest; a line passing through the longitudinal axis intersects obliquely the clavicle near its acromial extremity. The median line and the linea mammalis, are convenient landmarks for indicating the space which the heart occupies transversely. The median line divides the heart, leaving about one-third on the right and two-thirds on the left side. The left margin of the heart, in the male, extends to a point just within the nipple, which is situated on the fourth rib near the junction of the rib with its cartilage. The apex is about three inches to the left of the median line, and about an inch within the linea mammalis. The right margin extends from half an inch to an inch beyond the sternum on the right side. Viewing the several portions of the heart in relation to the median line, on the right are situated the right auricle and about a third of the right ventricle; on the left of this line are situated two-thirds of the right ventricle and the left auricle.

The relations of the heart to the adjacent organs are important with reference to the signs furnished by percussion and also by the other methods of exploration. At the base are the large arteries connected with the ventricles, viz., the aorta and pulmonary artery, which extend upward beneath the sternum, the latter to the level of the upper margin of the second, and the former nearly as high as the first rib. The course of these vessels, and their relations to each other, and to the thoracic walls, are of importance in regard to certain auscultatory signs, and will be referred to in that connection. The portion of the heart situated on the right of the median line is covered by the right lung.[1] The lower border of the heart, to the left of the median line, lies on the diaphragm which separates it from the left lobe of the liver, and, toward the apex, from the stomach. Its relations to the stomach are more or less extensive, according to the degree of distension of the latter organ. The portion of the heart lying to the left of the median line is only partially covered by the left lung; a part is in contact (the pericardium only intervening) with the thoracic walls. The space on the chest beneath which the heart is uncovered of lung, is called the *superficial cardiac region*. The præcordial space within which the heart is covered by lung, is called the *deep cardiac region*. These names will often recur, and their meaning should be understood. The left lung extends downward on the median line to the level of the junction of the fourth costal cartilage with the sternum. From this point the border of the lung diverges, leaving an irregular quadrangular portion of the heart's surface exposed. This space may be embraced with sufficient precision for practical purposes within a right-angled triangle, delineated as follows:[2] The oblique line, or hypothenuse, is drawn by connecting a point at the centre of the sternum on a level with the junction of the fourth costal cartilage, with the point where the apex of the heart comes in contact with the thoracic walls, the latter being usually in the fifth intercostal space, about an inch within the linea mammalis, or about three inches to the left of the median line. The median line extending from the same point on the sternum, and a line extending transversely from the point of the apex-beat to meet the median line, will form the two other sides of the triangle. The superficial cardiac region is thus bounded on two of its sides by lung, and on the greater part of one side, viz., the lower, by the liver and stomach, with the diaphragm intervening. The limits to which the deep cardiac region extends beyond those of the superficial cardiac region, have been already defined in giving the boundaries of the space which the heart occupies within the chest.

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[1] Vide Fig. 1, Frontispiece.

This account of the situation and anatomical relations of the heart, based on examinations of the dead subject, is sufficiently exact for practical purposes; but in the living body its relations to the thoracic parietes and the adjacent organs varies within certain limits, not only in different persons, but in the same person at different times. The size of the heart is variable, owing to a greater or less accumulation of blood in its cavities, more especially in the auricles. It is movable to some extent. The base is comparatively fixed, but the apex moves freely in a lateral direction, and varies in its situation in different postures of the body. The superficial cardiac region is larger or smaller according to differences in different persons as regards the volume of the left lung and the conformation of the chest. It is small in robust persons with deep chests, and larger in the slender and broad-chested. Its size is greater at the close of an expiration than after an inspiration, and the difference is, of course, marked in proportion as these respiratory acts are forced. These are variations irrespective of those occasioned by disease. Moreover, in the dead subject, the conditions of the heart and lungs affecting their mutual relations are by no means uniform. The lungs collapse and shrink away from the heart more or less, according to contingencies which are independent of disease; and the state of the heart, as regards the quantity of blood remaining in its cavities, depends on the mode of dying and other circumstances. But these variations are not sufficient to render unreliable the signs incident to diseases of the heart.

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[1] Vide Fig. 2, Frontispiece.

[2] Vide Fig. 1.

During life, the space within which the heart, in health, is uncovered of lung and in contact with the chest, in other words, the limits of the " superficial cardiac region," and the boundaries of the heart beyond these limits, or the " deep cardiac region," may be determined by means of percussion. With sufficient care and practice, the two regions just named, to the left of the median line, may be determined on the chest in the majority of persons. Their limits, in fact, are often so distinctly definable that, in view of the changes which occur in the heart and lungs after death, the dimensions obtained by percussion during life represent more fairly the normal relations of these organs than measurements with the parts exposed to view in the dead subject. The limits of the superficial cardiac region are best ascertained by light percussion, commencing at the centre of the region. The upper limit in seventeen healthy persons in whom it was carefully ascertained was the cartilage of the fourth rib; in some the upper and in others the lower margin of the cartilage near the sternum. The outer limit on a transverse line passing through the nipple is at a point varying from half an inch to an inch and a half within the nipple, the average distance in twenty-two persons being a small fraction over an inch. The apex-beat, which is generally either seen or felt, determines the outer limit at the base of the triangle. The percussion-sound at this point is sometimes tympanitic from transmitted gastric resonance, the quality and the pitch of sound denoting its source. In determining the lower boundary of the region, the line of demarcation between the liver and the lower border of the heart is to be distinguished by the percussion-sound. This is readily done in most persons. Percussing from a point over the liver toward the heart, namely, in the epigastrium in a direction upwards and outwards to the left, the flat, liver-sound, at a little distance above the xiphoid cartilage, gives place to dulness, as distinguished from flatness. Connecting now the several points, already marked on the chest with ink or some coloring substance, we have a diagram representing the superficial cardiac region sufficiently exact for ascertaining its normal dimensions in the living subject. The average transverse diameter, measured from the median line to the outer limit, a little below the level of the nipple, in twenty-three healthy persons, was a small fraction over three inches, the maximum width being four, and the minimum two and a half inches. The average vertical diameter, measured on the median line in sixteen healthy persons, was two and a half inches, the maximum being three, and the minimum one and three-quarter inches.

In determining the boundaries of the heart beyond the limits of the superficial cardiac region, that is, the extent of the "deep cardiac region," forcible percussion is requisite, but not force enough to occasion pain. In mapping on the chest this space, the course enjoined by Bouillaud has decided advantages, namely, commencing at some distance from the heart and percussing towards the præcordia. The points at which the percussion-sound changes, becoming slightly but distinctly dull, being marked and connected by lines, the space occupied by the heart is delineated on the chest; and if the limits of the superficial cardiac region are delineated on the same chest, we have two concentric diagrams representing the two regions. Attention to the pitch of the percussion-sound is of great assistance in appreciating the dulness within the deep cardiac region, a change in this respect being more readily recognized than the difference in the degree of resonance. Taking the nipple as a landmark, in twenty-five healthy persons (all males) the left border was precisely at the nipple in sixteen; in six instances, it was within the nipple, the greatest distance being seven-eighths of an inch, and the smallest three-eighths; in three instances it was without the nipple, being half an inch beyond in two, and three-eighths of an inch in the remaining instance.[1] The præcordial region, as determined by percussion on the living body, extends somewhat farther to the left of the sternum than when this region is viewed in the dead subject, a fact doubtless owing to the presence of a larger quantity of blood within the cavities of the left side of the heart during life. On the right side of the sternum, on a level with the nipple, dulness is generally appreciable within a space varying from half an inch to an inch. The percussion-sound over the third rib near the sternum is generally sufficiently changed on percussion from above downwards to indicate the base of the heart in this situation.[2]

The foregoing details, which have been given as succinctly as possible, are essential as constituting the basis of the physical signs of enlargement of the heart. The latter, after these preliminaries, may be briefly presented. The area of præcordial dulness exceeds the limits of health in proportion as the volume of the heart is abnormally increased. The effect of an enlarged heart is especially manifest in the superficial cardiac region. The heart, in proportion to its increase in volume, pushes aside the anterior border of the left lung, leaving a larger portion of

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[1] It should be stated that these, as well as the preceding results of percussion, were obtained by percussing while the persons were in a sitting posture.

[2] The combination of percussion and auscultation, or auscultatory percussion, as proposed and practised by Drs. Cammann and Clark, of New York, is undoubtedly well adapted to determine with ease and accuracy the boundaries of the heart. See New York Journal of Medicine, July, 1840. That this mode is not more generally employed is because percussion, as usually practised, suffices for ordinary practical purposes. The stethoscope recently invented by Dr. Cammann is well suited to auscultatory percussion. The publication by Drs. Cammann and Clark just referred to, contains the average dimensions of the space occupied by the adult heart in a series of examinations. The following are the mean results:

	Male.	Female.
Vertical diameter, . . .	4 in. 0 lines,	3 in. 7 lines.
Transverse " . . .	4 " 4 "	4 " 1 line.
Right oblique " . . .	4 " 10 "	4 " 10 lines.
Left oblique " . . .	3 " 10 "	3 " 7 "

its anterior surface uncovered and in contact with the thoracic walls. The superficial cardiac region, thus, becomes larger than in health. This effect is certainly the rule, and the exceptional instances, described by some writers,[1] in which the heart buries itself beneath the lungs, leaving its anterior surface covered to the same extent as in health, must be extremely rare, assuming the volume of the lungs to be normal. The apex is moved to the left of its normal situation, owing partly to the oblique position of the heart, and in part to the fixedness of the base of the organ, the latter, with the diaphragm below, offering mechanical resistance to much extension in a vertical direction. The apex being free, is moved readily in a lateral direction. The evidence, therefore, of the heart being abnormally uncovered of lung, and of the extent of its surface in contact with the chest, is obtained by percussing from the median line towards the nipple and towards the point where the apex-beat is felt. The lateral diameter of the superficial cardiac region at the inferior boundary, *i. e.*, between the median line and point of apex-beat, may be one, two, and even three inches greater than in health. The superficial dulness instead of ending an inch within a vertical line passing through the nipple, extends to this line, or an inch, two inches, or even more, beyond it. The presence of the apex-beat enables us to determine the diameter in this situation without practising percussion. The apex may be more or less lowered, as well as carried to the left. It is frequently found in the sixth or seventh, and it may be as low as the ninth, intercostal space, the inferior boundary of the superficial cardiac region being, of course, proportionately lower than in health. Percussing next from the left margin of the sternum on or just below the level of the nipple, the superficial dulness may be found to extend to the nipple, or half an inch, an inch, or even farther, beyond it. The diameter of the region here will correspond to the abnormal width of the heart. Other things being equal, the enlargement of the heart transversely may be accurately measured by the extent to which the diameter of the superficial cardiac region in this situation is increased. But it is to be borne in mind that the normal situation of the outer limit of this region is not the same in all persons. The average distance within the nipple is very nearly an inch, but the variation within the range of health, as has been seen, is from half an inch to an inch and a half. If the superficial dulness extend to within half an inch of the nipple, or possibly even within a still shorter distance, it may not be due to abnormal enlargement; and, on the other hand, in a person whose heart is normally covered by lung an inch and a half within the nipple, superficial dulness extending to a point within half an inch of the nipple would denote considerable enlargement of the heart. If the area of superficial dulness proper to the individual be not known, an abnormal increase of its dimensions cannot in any case be assumed unless the lateral diameter extend nearly or quite to the nipple. Here, as in other instances, the extreme limits of healthy variation are of greater practical consequence than averages. In determining, however, whether the heart be enlarged or not, the distance from the apex to the median line is to be taken into account, and also the signs obtained by other methods of exploration than percussion.

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[1] Traité de Diagnostic, par Racle.

The degree of dulness within the superficial cardiac region is greater than in health in proportion to the enlargement. In health, a portion of the heart is imbedded in lung sufficiently to occasion the transmission of more or less pulmonary resonance over the whole of the præcordia. The degree of normal dulness differs in different persons. It is generally marked, and sometimes approaches to flatness. It is sufficient to render the limits of the region distinctly definable, except when great obesity exists, or, in the female, when the mammary development is unusually large. It is sufficiently intelligible that, in proportion as the lung is pushed aside in cases of enlargement, the dulness will be greater in degree than in health. In some instances it amounts to flatness. It is equally obvious that the sense of resistance felt in practising percussion will be marked according to the increased bulk of the heart.

It is important to bear in mind that increased extent and degree of superficial dulness are signs of enlargement of the heart, with this provision, namely, that the lungs are free from disease. The size of the area is affected by abnormal conditions of the latter organs, as well as the heart. In phthisis, the left lung is frequently contracted, so that the anterior margin is removed towards the border of the heart, leaving a larger border of the heart's surface in contact with the thoracic walls, even though the size of the organ may be less than in health. A similar result follows chronic pleurisy, the lung not expanding and resuming its normal volume sufficiently to cover the heart as in health. In these cases the liability to error is slight, for the existence of tuberculosis is determined without difficulty, and the retrospective diagnosis of pleurisy is also easily made; moreover, the præcordia is not enlarged, and all doubt is removed by defining the boundaries of the deep cardiac region.

The relations of the heart and lungs are also affected by a variety of causes, irrespective of morbid conditions of either of these organs, such as enlargement of the liver, dilatation of the stomach, aneurism of the aorta, enlarged spleen, ascites, pregnancy, tumors in the mediastinum, &c. These disturbing causes are generally determinable; and the importance of not limiting exploration to the præcordia, but extending the examination over the chest and abdomen in order to exclude any affections which alter the normal relations of the heart and lungs, is sufficiently obvious. Errors of diagnosis are sometimes attributable to neglect of this precaution.

The limits of deep dulness are not extended beyond those of superficial dulness proportionately to the degree of enlargement of the heart, but it is sometimes desirable to ascertain the actual space which the heart occupies. Percussing from without the heart toward the præcordia, the lateral borders of the organ may generally be determined without great difficulty. The enlargement of the deep cardiac region is manifested by dulness extending more or less without the left nipple, and in some cases, also, beyond its normal boundary to the right of the sternum. Not only the extent of this region, but the form of the heart may be delineated, and the latter is of diagnostic significance as respects the discrimination between hypertrophy and dilatation, the latter increasing more than the former the width in proportion to the length of the heart.

The evidence afforded by percussion of enlargement of the heart is much less marked, if, in conjunction, the left lung be affected with emphysema. This combination is not infrequent. The effect of emphysema of the left lung is to lessen and even abolish the superficial cardiac region. The anterior border of the lung may be extended forward so that the whole surface of the heart is covered. The heart, too, is often depressed below its normal situation by the pressure of the dilated lung. The coexistence of emphysema, thus, renders the area of the superficial cardiac region no longer an index of the existence and the degree of enlargement of the heart. The limits of the deep cardiac region are alone to be depended on, and they are not always, under these circumstances, easily defined. The combination renders the diagnosis difficult by impairing also concurrent signs of enlargement obtained by auscultation, inspection, and palpation. Moreover, the symptoms of emphysema are liable to be confounded with those which are due to disease of heart. The individual cases in which this difficulty in diagnosis exists are easily recognized, for the signs of emphysema are sufficiently explicit; and in a certain proportion of these cases we must be content to rely in a great measure on the well-known pathological association of the two affections, determining the relative proportion of each approximatively.

Enlargement of the heart results from different pathological conditions. In addition to the two forms, to the consideration of which this chapter is devoted, viz., hypertrophy and dilatation, the organ acquires an abnormal size from the accumulation of blood within its cavities and the deposit of morbid products and fat on its surface. The question may be here raised, whether percussion furnishes data for the differential diagnosis of the different varieties of enlargement. Hypertrophy or dilatation, as has been seen, may be limited to portions of the heart, or may disproportionably affect certain portions. It is stated that the dulness extends more to the left of the median line when the left ventricle is the seat of enlargement, and more to the right of this line when the right ventricle is affected. The relations of the two ventricles, however, are such that, in view of the position of the heart and the movableness of the body and apex, the left border is extended in proportion as the right side is increased in size, and, as a rule, the foregoing statement does not hold good clinically. The right or left auricle, belonging to the base which is comparatively fixed, when considerably enlarged, may occasion a greater relative extent of dulness on the corresponding side of the sternum. Great distension of the right side of the heart, may be manifested by an abnormal extent of dulness over the site of the right auricle; and this extent of dulness may be found to have diminished when the causes of obstruction are removed. The ability to distinguish between hypertrophy and dilatation by the percussion-sound is more than questionable. This is a nicety which the student should not attempt to acquire, for in proportion as he might imagine that he had made the acquisition, would be his liability to error in practically trusting to it. The same remark is applicable to the endeavor to determine by percussion that enlargement of the heart is due to the deposit of fat or morbid products on its surface. Very considerable extension in a transverse direction of the superficial and deep cardiac regions, is presumptive evidence that the increased volume is due to dilatation rather than hypertrophy, for the former, more than the latter, tends to increase the width of the organ and also to give rise to excessive augmentation of size. Extension in this, rather than in a vertical, direction, is evidence of enlargement of the right, instead of the left, ventricle. On the other hand, if percussion show that the heart is considerably lengthened, and that the transverse enlargement is not disproportionate to the vertical, the presumption is in favor of hypertrophy rather than dilatation, and of hypertrophy affecting chiefly the left ventricle.

Enlarged extent and degree of præcordial dulness are produced by liquid accumulation within the pericardial sac, as well as by enlargement of the heart. Both may coexist, and then the evidence afforded by percussion of enlargement of the heart ceases to be available. The points of distinction between the præcordial dulness or flatness due to liquid accumulation within the pericardial sac, and that due to enlargement of the heart, are important, and will be considered in connection with the subject of pericarditis.

2. *Altered situation and extent of the apex beat; impulses elsewhere than over the apex of the heart, and abnormal force of impulse, as determined by palpation.*

The point at which the apex comes into contact with the thoracic walls, is in the fifth intercostal space, the person examined being in the sitting posture. Of twenty-five healthy persons examined, none presented an exception to this rule. In this intercostal space, the impulse is felt over an area varying from half an inch to an inch and a half. in health. The average transverse diameter of this area, in thirteen persons, was a fraction over an inch. The centre of this area, where the beat is strongest, is situated within the linea mammalis, at a distance from that line varying from two inches to three-eighths of an inch, the average, in eighteen persons, being a fraction over an inch. The distance from the median line to the centre of this area varies between three inches and five-eighths and two inches and five-eighths, the average, in fifteen persons, being a fraction under three inches. Measured from a transverse line passing through the nipple, the distance varies from an inch and an eighth to two inches, the average, in eight persons, being a fraction over one and a half inch. These are the relations of the apex-beat in the sitting posture. Deviations take place when the posture is changed, owing to the movableness of the apex of the heart. In the recumbent position on the back, the beat is frequently felt in the fourth intercostal space, the same relations laterally to the nipple and median line as in the sitting posture being preserved. The frequency with which this is observed has led some writers to state, incorrectly, that, as a rule, the apex-beat is in the fourth intercostal space.[1] Lying on the right side, the centre of the area within which the beat is felt is moved about half an inch nearer the sternum. Lying on the left side, the beat is moved to the left, so that the centre of the area generally falls on the linea mammalis, and the impulse is felt half an inch without this line. The respiratory movements sometimes affect the situation of the apex-beat. I have not observed it to be lowered by a full inspiration, but it is occasionally raised from the fifth to the fourth intercostal space by a forced expiration, the persons being examined in the sitting posture. The apex-beat is not infrequently inappreciable to the touch in healthy persons, in the sitting posture. The persons in whom it is wanting have generally deep chests. Thickness of the soft parts also prevents it from being felt. It is lost in the recumbent position on the back in some instances in which it is felt when the person is sitting. It is still oftener lost when the person lies on the right side, but very rarely when the position is on the left side. In the latter position it is sometimes felt when it is not appreciable in any other.

The force of the impulse varies in different persons. It is rarely strong when the person is tranquil and free from mental agitation. It is almost invariably weaker when the person lies on the back than in the sitting posture; and it is still more diminished, if not lost, when the position is on the right side. Lying on the left side increases the impulsive force; the beat is strongest in this position. The sensation on applying the fingers over the area of the apex-beat, as stated by Walshe, is that of a gliding as well as an impulsive movement. It is not that of a percussion or blow. Of course, the apex does not withdraw itself from the thoracic walls, and then come into contact through an open space. The beat must, of necessity, be produced by movements incident to the changes in form of the organ, and not to the tilting forward of the apex, as was formerly imagined.[1]

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[1] Verneuil, 1852, Racle, op. cit.

Directing attention now to the signs of enlargement and of hypertrophy obtained by palpation, those relating to the situation of the apex-beat are to be first noticed. The apex-beat is carried to the left of its normal situation and frequently lowered when the volume of the heart is increased. These changes are among the most constant and reliable of the signs of enlargement. The beat may be felt one, two, three, or more inches without the nipple. It may be found in the sixth, seventh, or eighth intercostal space. The distance to which it is moved in these directions, assuming that the alterations depend exclusively on the increased volume of the heart, constitutes a criterion for estimating the amount of enlargement. It must, however, be considered that abnormal conditions extrinsic to the heart alter the relations of the apex to the walls of the chest, such as enlargement of the left lobe of the liver, distension of the stomach, ascites, and enlarged spleen; these and others pertaining to the abdomen may move the apex to the left, but without lowering it. An aneurismal or other tumor situated above the heart may cause it to be moved more or less to the left of its normal situation and also downward. An emphysematous left lung pushes the heart downward and toward the epigastrium, often giving rise to an impulse in this situation, while the normal apex-beat is suppressed. These extrinsic conditions are, of course, to be excluded before the abnormal situation of the apex can be regarded as a sign of enlargement of the heart.

The lowering of the apex-beat and the distance to which it is carried to the left are not uniformly in equal proportion; that is, the apex-beat is in some cases lowered to a much greater extent that it is moved to the left, and *vice versa*. As a rule, it may be said that the enlargement is great in proportion to the extent to which the apex is either lowered or moved to the left, always excluding causes affecting its situation which are extrinsic to the heart. The apex, in some cases, is carried far to the left, and upward instead of being lowered. It may be found nearly in the axilla. This probably does not occur except when the apex is pushed upward by some of the extrinsic morbid conditions just named.

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[1] The consideration of the mechanism of the heart's beat, to which some space was accorded in the first edition of this work, is omitted in this edition. For this the reader is referred to treatises on physiology.

The area in which the apex-beat is felt (averaging about an inch in health) is enlarged in cases of enlargement of the heart. The apex is less pointed than in health; it is blunt or rounded, and consequently a broader surface comes into contact with the thoracic walls during the systolic impulse. This is a sign of some importance taken in connection with other signs denoting enlargement either by hypertrophy or dilatation.

In cases of hypertrophy of the left ventricle, the force of the apex-beat is abnormally great in proportion to the increased thickness of the walls, provided that the form of the apex be not much altered, or the completeness of the ventricular contractions not prevented by contraction at the aortic orifice, or other causes. An abnormal force of the apex-beat is always associated with change in situation and extension of the area in which the beat is felt. The force of the beat is an important sign as showing that the enlargement is due to hypertrophy rather than to dilatation, or that the former predominates. In proportion as the left ventricle is hypertrophied, rather than dilated, other things being equal, the force of the beat is increased. An increase of the force of the beat, however, may be due simply to increased muscular activity of the organ without enlargement, the heart being affected functionally or dynamically without organic disease. The beat is increased in the same manner as under excitement by active exercise or from mental agitation. How is it to be determined whether the abnormal force of the beat be due to hypertrophy or simply to morbid excitement or palpitation? The sensation in the latter case is that of increased action, and in the former case of increased power. The beat in hypertrophy is felt to be produced by a strong contraction of the ventricle; the impression conveyed by the touch is that of a prolonged, as well as a strong impulse. In purely functional excitation, the beat is more abrupt, quick, and brief, giving the idea of violence rather than of strength.

The distinction is important, and would be vastly more so were the discrimination to rest solely on the difference, as respects the force of the beat. But in hypertrophy there are the coexisting signs of enlargement which are wanting in an affection simply functional. Increase of the power of the apex-beat, however, is not a constant sign in cases of hypertrophy. Owing to the globular form of the heart and its rounded or blunted extremity, the apex in some cases does not make strong pressure against the walls of the chest. Moreover, the situation of the apex may be such that the pressure is against a rib and not in an intercostal space. For these reasons, the apex-beat may be feeble, notwithstanding the existence of hypertrophy. These reasons account for a feeble apex-beat especially when hypertrophy is limited to the right ventricle. It is important to bear in mind the feebleness of the apex-beat, in some cases of enlargement by hypertrophy, lest it be overlooked, and a stronger impulse in the intercostal space above be considered as indicating the situation of the apex. This error would be likely to lead to a false conclusion as regards the amount of enlargement of the heart. The lowest impulse, whether strong or feeble, is, of course, the apex-beat.

Abnormal impulses are often felt above the apex. Occasionally, in health, in addition to the apex-beat in the normal situation, an impulse is appreciable in the fourth intercostal space, and, in some instances, in the epigastrium to the left of the xiphoid cartilage. In cases of enlargement, impulses may be felt in three, four, or even more intercostal spaces, and sometimes in each space between the base and apex. In these cases the lowest point of impulse is the farthest removed from the median line, and each of the above impulses is nearer the sternum. The impulses above the apex, as just stated, may be stronger than the apex-beat. Not infrequently the latter is weak, and perhaps scarcely appreciable, while the former are notably strong. The explanation of these additional impulses is, the heart being in contact with the thoracic walls over a larger space than in health, in other words, the area of the superficial cardiac region being enlarged, the movements of the organ are communicated to the yielding spaces between the ribs. This does not take place, as a rule, in health, in consequence of the interposition of lung save over a comparatively limited space. The impulsive movements, elsewhere than over the apex, are not always coincident with the ventricular systole; in other words, the elevations or outward motions at the several points at which the movements are observed, do not take place in unison, but in some instances in alternation. Thus when movements are felt in the fourth and sixth intercostal spaces, that in the sixth is the apex-beat and systolic, while that in the fourth may occur with the diastole of the ventricles. Alternation of the impulsive movements in these two intercostal spaces, is not infrequently observed in cases of hypertrophy. The superior or diastolic movement was called by Hope the back-stroke of the heart. It is stated that this is sometimes observed in healthy persons when the heart acts with unusual vigor.[1] Generally in the cases in which a diastolic movement is observed, retraction of the intercostal space takes place during the ventricular systole, due to the flattening of a portion of the heart, and the movement of impulsion which alternates with the apex-beat is, in fact, only the elevation of the space to the level from which it was depressed. In other words, the space over the body of the heart yields to atmospherical pressure and follows the retreating ventricular walls during the systole, resuming its level when the heart assumes a more globular form during the diastole. The diastolic impulsion is not strong, and may be visible when not distinctly felt. An impulse over, or a little below the base of the heart, *i. e.*, in the third intercostal space, is referable to the expansion of the upper portion of the organ during the systole. The fact of this expansion and the force with which it takes place are shown by grasping the heart near the base in a living animal; a strong pressure is felt when the ventricles contract. It is not difficult to understand that the change of form at the base should communicate an impulsive movement to the intercostal space when the heart is abnormally uncovered of lung, and also in some instances of palpitation without organic disease, when the action of the heart is notably augmented. In some instances, the dilatation of the pulmonary artery during the systole of the ventricles, or the shock produced by the sudden arrest of the column of blood in consequence of the expansion of the sigmoid valves during the ventricular diastole, may give rise to an impulsive movement which may be felt in the second left intercostal space. An impulse is sometimes felt in this situation when, from pulmonary disease, the left lung recedes at this point, leaving the artery uncovered and in contact with the parietes of the chest, the heart being free from disease. An impulse situated here, referable to the pulmonary artery, is more likely to occur, for obvious reasons, in cases of hypertrophy of the right ventricle and when there exists obstruction to the pulmonary circulation. Laennec supposed that an impulse on the left side at the base of the heart was sometimes due to the contraction of the left auricle. Aside from the fact that the greater part of the auricle is covered by the large arteries emerging from the heart, and the improbability of its ever contracting with sufficient force to communicate a perceptible impulse to the walls of the chest, it is difficult to understand how any other than a movement of retraction can accompany its systole. It is more reasonable to attribute an impulse in this situation, either to the ventricles or to the pulmonary artery. If there be free regurgitation through the mitral orifice, it is intelligible that the retrograde current of blood impelled by the force of the systole of the left ventricle may occasion an impulse over the auricle. This is perhaps the explanation in some instances, at least, in which an auricular impulse has been supposed to exist. Stokes has observed a case in which an impulse was felt on the right side of the sternum, evidently, from the appearances after death, due to a retro-current through the tricuspid orifice, the right auricle being enormously dilated and its walls attenuated.[1] It is indeed possible that without insufficiency of the mitral or tricuspid valves, an impulse may be produced by the momentum communicated to the blood contained within a dilated and distended auricle by the backward pressure of these valves during the ventricular systole.

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[1] Bellingham on Diseases of the Heart. Dublin, 1853. Part I, p. 81.

It is to be borne in mind that the movements in intercostal spaces, impulsive or retractive, involve contingencies which are irrespective of cardiac disease. They are more likely to occur in persons who have flattened chests and long sternums than in those with a thoracic conformation the opposite of this. They require a certain thinness of the parietes of the chest, and are more marked in proportion as the thoracic walls are attenuated. They may be obvious at times when the heart is unusually excited and not appreciable when the organ acts more quietly. They may be due to abnormal conditions pertaining to the lungs, the heart remaining sound. They are observed in some instances in which the pulmonary substance is withdrawn from the heart, as after the absorption of liquid effusion in pleurisy affecting the left side, and in some cases of tuberculosis. An effect of these affections is often to leave an enlarged area of the heart's surface in contact with the walls of the chest, and, under these circumstances, the motions of the organ may communicate corresponding movements to the intercostal spaces. Hence, impulsive movements elsewhere than over the apex of the heart are never signs of enlargement, unless associated with altered situation of the apex-beat and other signs indicating that the volume of the organ is increased.

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[1] On Diseases of the Heart and Aorta, Am. ed., p. 290. Dr. Stokes attributes the impulse over the dilated auricle, in that case, to the auricular contraction; but as he states that it was synchronous with the ventricular systole or the first sound of the heart, it seems clearly to have been due to a regurgitant current.

The conformation of the chest in some persons is such that an impulse referable to the heart is felt, in health, in the epigastrium by directing the fingers upward beneath the false ribs on the left side. In the majority of persons the action of the heart is not appreciable in this situation. Cardiac impulse in the epigastrium is therefore usually, but not invariably, a sign of disease. As a morbid sign, it denotes either enlargement of the heart or displacement in a downward direction. It is a sign by no means present in most cases of enlargement of the heart. The oblique position of the heart and the resistance offered by the diaphragm and the left lobe of the liver prevent much descent towards the epigastrium. These circumstances apply, as has been seen, measurably, to cases in which the enlargement predominates in the right as well as the left ventricle. But it is undoubtedly true that an impulse in this situation is more likely to occur as a result of enlargement of the right than ot the left ventricle. When it proceeds from a cardiac affection, it may be considered as affording strong presumptive evidence that the right ventricle is enlarged. A strong impulse, under these circumstances, goes to show that the enlargement involves not merely dilatation, but hypertrophy. The question to be first settled is, Does it proceed from increased size of the heart? This question may be settled frequently by reference to the apex-beat. If the beat be in its normal situation, and there be no signs of enlargement, the impulse in the epigastrium is probably normal; it is not a sign of disease. But if the apex-beat be removed to the left of its normal position, it becomes a sign of enlargement of the right ventricle. When this is the case, other signs of enlargement will also be present. The diagnostic value of the sign, thus, when it is attributable to a cardiac affection, consists in its indicating that the right ventricle is the seat of enlargement. When it is determinable that the epigastric impulse is due to cardiac enlargement, the extent of the impulse will, in some measure, be an index of the amount of enlargement of the right ventricle, and the force of the impulse will be in proportion as the enlargement is by hypertrophy. The impulse is communicated in some instances not only to the epigastrium, but to the lower part of the sternum, and it is sometimes sufficient to cause a movement, perceptible to the eye and touch, which extends over the site of the liver. When due to displacement of the heart, in the great majority of instances it is dependent on emphysema affecting the left lung. The dilated lung presses the heart downward, overcoming the resistance offered by the diaphragm and liver, and the action of the right ventricle is felt in the epigastrium. The signs and symptoms of emphysema are sufficient to establish the fact that this cause of displacement exists. The apex-beat, under these circumstances, is frequently or generally wanting. Emphysema, however, induces enlargement of the heart, seated primarily and especially in the right ventricle. The epigastric impulse, therefore, may be due to both causes combined, viz., enlargement and displacement. To determine the proportion which each bears in the production of the sign is not always easy; but if the boundaries of the heart be determinable by percussion, or if the situation of the apex-beat can be ascertained, this point may be settled with much precision. In examining the epigastrium with reference to the evidence of enlargement of the heart, it is important not to confound an impulse referable to the heart with a pulsation often felt in that situation which is only indirectly attributable to the heart's action. In some thin persons, the beating of the descending aorta may be felt here; and in connection with hysteria and other nervous affections, a strong arterial pulsation is perceived in the epigastrium. It is generally not difficult by means of palpation either to trace these pulsations directly to the heart or to isolate them from the latter.

The action of the heart is frequently attended by a shock felt by the hand or the head applied over the præcordia. Sensible movements are also sometimes communicated to the ribs, as well as the intercostal spaces, and they may extend over the præcordia. A perceptible and more or less forcible shock attends certain palpitations which are merely functional. The heart appears to act with violence. It seems to knock against the ribs. The sensation, in some instances, is as if the chest were struck with a hammer. The patient is painfully sensible of the force of the impulsion, whereas, in health, if the heart be not excited, its movements take place without the mind being cognizant of them. The violence of the action is shown by the movements of the body, the dress, or the bedclothes. The instances related of fracture of the ribs and detachment of the costal cartilages by the force of the heart's action are doubtless apocryphal, but the shock is sometimes notably violent. It may be limited to the apex, or it may be felt at the base, and, indeed, over the whole præcordia. Alone, the shock, however violent, only indicates excited action of the heart. It does not, of necessity, imply organic disease. It may be due simply to the fact that the heart acts with spasmodic quickness and force. It is represented by the intensity of action incident to fear and other emotions. If it be inorganic or functional, it is usually temporary, unattended by physical signs denoting organic lesions, and characterized by circumstances which will be hereafter considered as distinctive of nervous disorder or palpitation. Organic disease, it is true, is often attended by violent action of the heart, but the significance of the latter as a sign of the former depends on the coexistence of other signs which are more unequivocal. A strong heaving movement of the præcordia is, however, diagnostic of enlargement by hypertrophy. This is quite different from the shock which has just been described. It is a comparatively prolonged, powerful elevation of the thoracic walls. The head, applied as in immediate auscultation, is raised, and, by the hand placed over the præcordia, the heart is felt to act with abnormal strength. The shock, due to intense functional excitement, proceeds merely from exaggerated action of the heart; the heaving movement in hypertrophy involves, in addition, increased power of the muscular contractions of the organ. Moreover, in the latter case, the surface of the heart being in contact with the thoracic walls over a larger area, the extent of the impulsive movement is greater. The distinction just made is the same as has been already pointed out in contrasting the prolonged, powerful apex-beat of a hypertrophied heart with the smart, sharp, violent impulse which indicates excited activity of the ventricular systole. The distinction in both instances is practically important, but in discriminating between functional disorder and organic disease, in practice, the diagnostician will, of course, be guided by the absence or concurrence of other signs. It is to be added that heaving of the præcordia is not uniformly present in hypertrophy. The presence of this sign involves, as a condition, a degree of functional activity proportionate to the augmented thickness of the ventricular walls; in other words, it will not be present if the muscular power of the heart be weakened from any cause, notwithstanding the augmented volume of the organ. While, therefore, the presence of this sign is evidence of the existence of hypertrophy, its absence is not positive proof that hypertrophy does not exist.

3. *Abnormal modifications of the heart-sounds; diminished extent and degree of the respiratory murmur and of vocal resonance within the præcordia, as determined by auscultation.*

The clinical importance of abnormal modifications of the heart-sounds has relation more to valvular affections than to enlargement of the heart. They are, however, by no means unimportant in connection with the latter. And here it will be necessary to premise some account of the heart-sounds in health. To enter into a discussion of the numerous theories which have been advanced with regard to the mechanism of these sounds, would be foreign to the practical character of this work. I shall limit myself to a concise statement of certain points which are essential as preliminary to the study of the phenomena of disease.

In studying the heart-sounds the stethoscope should be used, in addition to immediate auscultation. The stethoscope enables the auscultator to study sounds from within a circumscribed space, whereas, with the ear applied to the præcordia, the sounds from different sources are commingled, and they cannot be so well studied separately. The binaural stethoscope, as devised by the late Dr. Cammann, is vastly superior to other stethoscopes in auscultating the sounds from the heart as well as from the lungs. For those who are not accustomed to this instrument it is important to add that a certain amount of use is necessary in order to appreciate its advantages, and that the instrument used should be properly constructed. Many are disappointed in making trial of it because certain nice points in its construction, which are essential, have not been attended to. The proper direction of its curves, and the size of the knobs which are introduced into the ear are especially important. A fair trial of it involves its use for several weeks at least.[1]

The two heart-sounds which together form the beat or revolution of the heart, are called the first and second, or the systolic and diastolic sound. By the latter terms, it is implied that the first sound occurs during the systole and the second sound during the diastole of the ventricles. This may be assumed as sufficiently established. These sounds, respectively, have their maximum of intensity, and their characters are best studied, in different situations, namely, the first sound over the point where the apex-beat is felt, and the second sound just above the base of the heart, in the intercostal space between the second and third ribs close to the sternum. Studied in these different situations, the two sounds differ as respects duration, pitch, and quality. The first sound, over the apex, is longer, lower, and has a booming[2] quality. The second sound, in the second intercostal space near the sternum on either side, is shorter, more acute, and has a clicking or valvular quality. These differences between the two sounds are generally well marked when the comparison is made in the different situations mentioned, but, as will be seen presently, they are much less marked in other situations within the præcordia. The sources of each of the sounds, and the parts concerned in its production, are important to be considered. With reference to these and other points, we will notice each sound separately, commencing with the second sound.

The second sound succeeds the first after an interval extremely brief, but, when the beats of the heart are not accelerated, distinctly appreciable. This sound, *i. e.*, the second, as already stated, is best studied just above the base of the heart, in the space between the second and the third rib, close to the sternum. If the second sound be compared on the two sides of the sternum, a difference in pitch and other characters is generally apparent. On the right side the sound is more acute, more abrupt, louder, and apparently nearer the ear. These differences, taken in connection with the anatomical relations of the aorta and pulmonary artery in these situations, and also with clinical facts pertaining to disease, warrant the conclusion that, when a disparity exists, the sound on the left side emanates from the pulmonary artery, and that on the right side is from the aorta. The sound in both situations has an unmixed, valvular quality, and, in view of the results of experiments made on living animals, and the effects of disease, it may be assumed that the valves of the aorta and pulmonary artery are the parts immediately concerned in its production. There is, then, a pulmonary second sound, due to the expansion of the valves of the pulmonary artery succeeding the ventricular systole, and an aortic second sound, referable to the semilunar valves of the aorta. The second sound of the heart presents the characters of that due to the pulmonary valves at the inferior border of the organ, *i. e.*, just above the xiphoid cartilage in some persons; occasionally, also, in the third intercostal space on the left side and over the body of the heart, within the superficial cardiac region. Elsewhere, within the præcordia, and at points removed from the præcordia, wherever the second sound is heard, it presents the characters distinctive of the sound produced at the aortic orifice. These facts are ascertained by comparing, in a sufficient number of healthy persons, the second sound, as heard at different points, with that heard in the second intercostal space on the right and left side. It follows from the facts just stated that the aortic second sound is much more intense and widely diffused than the pulmonary, the latter, in some persons, being distinguishable alone in the second intercostal space on the left side; sometimes, indeed, the aortic sound predominates even in that situation. The second sound of the heart maintains its distinctive characters of pitch, duration, and valvular quality, unaffected by the causes which affect the movements of the heart within the limits of health, such as exercise, mental agitation, &c. Its intensity even is not much affected by these causes. These facts show its unmixed character, in other words, that it consists of a single element only, a valvular element, in this respect differing from the first sound of the heart.

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[1] Dr. Frederick G. Snelling, of New York, has suggested a useful addition to the binaural stethoscope, namely, a rim of India-rubber extending about half an inch beyond the pectoral extremity. By means of this addition, the application may be made more easily in emaciated patients, discomfort from the pressure is avoided, and the conduction is somewhat increased.

[2] The term booming, borrowed from Walshe, has not a very definite signification; but expresses a difference in quality difficult to be described, although easily appreciated by the ear.

The first sound of the heart, studied at the situation where its intensity is greatest, viz., over the apex of the organ, is a mixed sound. In this situation, it is said to be *accentuated*, that is, in the succession of the two sounds the stress falls upon the first, while at the base of the heart, and at other points, the accent is on the second sound. This, however, is only saying that over the apex the first sound is louder than the second, and at the base of the heart the second is louder than the first sound. The mixed nature of the first sound is shown by the difference which it presents on auscultation over the apex, and at other points within the præcordia; by contrasting its characters as heard when the stethoscope is firmly placed directly on the surface of the chest with those which it presents when several thicknesses of some soft material are interposed between the instrument and integument, or when the instrument is imperfectly applied; by auscultating over the apex when the person examined is placed in different positions, and taking into consideration modifications incident to certain diseases and peculiar to certain persons in health.[1] The clinical study of this sound in health and disease leads to the conclusion that it is composed chiefly of two different elements. One of these elements consists of a clicking sound, emanating from the mitral and tricuspid valves. The other element, in the author's opinion, proceeds from the movements of the apex of the heart against the thoracic walls. In a practical point of view, however, it is unimportant whether the latter element be thus explained or whether it be accounted for on the hypothesis of a sound caused directly by muscular contraction. Referring it to the movements of the apex against the thoracic walls, this element may be called the *element of impulsion*, and the other element the *valvular element*.[2] These names will be employed in this work to distinguish from each other the two elements composing the first sound. Of the fact of the maximum of intensity of the first sound being at the apex, the following is an explanation: The right ventricle is in contact with the base of the chest within the superficial cardiac space except at the apex; hence, the valvular sound produced at the mitral orifice, conducted by the papillary muscles, is loudest at the apex. The sound due to impulsion is loudest in this situation because it is here produced.

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[1] Vide "On the Clinical Study of the Heart-sounds in Health and Disease" (prize essay by the author), Trans. Am. Med. Association, 1858. This essay contains a full account of the results of the clinical study of this sound under the different circumstances mentioned above.

[2] Discussion of this opinion, respecting the mechanism of the element of impulsion of the first sound, is waived in this work. The reader is referred to the author's prize essay on the clinical study of the heart-sounds for the grounds on which the opinion is entertained. I will simply add here that the experiment of placing Cammann's stethoscope over the naked heart, when exposed in a living animal, seems to me sufficient to disprove the hypothesis that muscular contraction furnishes an element of the first sound. The first sound in this experiment

The two elements of the first sound are combined in different proportions in different persons in health; also, in different situations in which auscultation is practised, in different positions of the body, as well as under different circumstances pertaining to disease. At certain points, the element of impulsion may be eliminated, leaving the valvular element alone present. The element of impulsion predominates and drowns the valvular element, often, on auscultation over the apex. It predominates, as a rule, over the body of the heart. At the base of the heart the valvular element generally predominates. At the left border of the heart, over the left nipple, the valvular element predominates, and, on carrying the stethoscope to the left of this point for a certain distance, the element of impulsion is eliminated, and the valvular element remains, leaving the sound as purely valvular in quality and as short as the second sound. The valvular element predominates generally at the right border of the heart and at all the points removed from the præcordial region where the first sound is appreciable. These facts, established by the clinical study of the heart-sounds in health, show that, although the element of impulsion predominates over the apex and body, only the valvular element is much diffused beyond the limits of the organ. The valvular element is less intense than the second sound, the latter being often heard in situations to which the former is not transmitted, viz., on the lateral surfaces of the chest, in the right infra-clavicular region, and over the back. The explanation of the fact that the valvular element of the first sound is generally weaker than the second sound, notwithstanding the auricular are so much larger than the semi-

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is intensely valvular. This sound sometimes has a similar intense valvular quality, in cases of great functional excitement of the organ, when the stethoscope is applied on the chest over the point of apex-beat, the element of impulsion being, from some cause, wanting.

lunar valves, is as follows: The mitral and tricuspid curtains, when the systole of the ventricles takes place, are floated out and in apposition to each other in consequence of the ventricles being filled with blood. The effect of the ventricular systole, therefore, is simply to make the curtains suddenly and forcibly tense. On the other hand, the aortic and pulmonic valves, resting in contact with the walls of the arteries, are expanded, as well as made tense, by the recoil of these arteries on the columns of blood. The difference in size between the auricular curtains and the semilunar valves explains the lowness of the pitch of the valvular element of the first sound, and the higher pitch of the second sound of the heart.

The relative intensity of the valvular element and the element of impulsion, in the first sound of the heart, varies considerably in different healthy persons. The variation is still greater in cases of disease. The element of impulsion is marked in proportion to the power of the ventricular systole, and the extent of the movements of the apex in contact with the thoracic parietes. The intensity of the valvular element is also affected by the ventricular systole; but another important condition is involved, namely, the quantity of blood within the ventricles at the time of their contraction. If the ventricles are well filled with blood, the valves are nearly or quite closed when the ventricular systole takes place. The valvular tension then yields a feeble sound. The valvular element may, indeed, be wanting if the ventricles be distended with blood in some cases of disease. On the other hand, in proportion as the quantity of blood is small when the ventricular systole takes place, the range of movement of the valvular curtains is greater, and, other things being equal, the intensity of the valvular element is increased. Different affections thus increase or diminish the intensity of the valvular element accordingly as the quantity of blood in the ventricular cavities is increased or diminished.

The valvular element of the first sound, as stated already, is due to the action of the mitral and tricuspid valves. Is the sound emanating from each of these valves ever distinguishable from the other? Clinical observation warrants an affirmative answer to this inquiry. Over the inferior border of the heart, near the xiphoid cartilage, this element frequently differs in pitch from the same element when heard in the same person at or without the left nipple. This may be considered as sufficient to render it at least highly probable that the source of the sound in the latter situation is at the mitral, and in the former situation at the tricuspid valves.

A striking point of disparity between the first and second sound of the heart relates to the extent of variation in intensity in different persons, and in the same person under different circumstances, within the limits of health, as well as in connection with disease. The first sound varies considerably in intensity according to the energy with which the heart contracts, and according to the posture assumed; it is often feeble when the person lies on the back as compared with intensity in the sitting posture, or lying on the left side. The second sound, on the other hand, undergoes little change in intensity under these and other circumstances, irrespective of morbid conditions. The variation to which the first sound is liable relates chiefly to the element of impulsion. The valvular element, like the second sound of the heart, is not subject to as much variation in intensity, exclusive of disease.

The relatively greater length of the first sound of the heart, as compared with the second sound, depends on the element of impulsion. In proportion as this element is predominant, the sound is prolonged; and, on the other hand, whenever this element is eliminated, the first sound is no longer than the second. The interval between the first and second sound is determined by the length of the first sound. This interval is shortened in proportion as the first sound is prolonged, and it is lengthened in proportion as the element of impulsion of the first sound is impaired or eliminated.[1]

The foregoing brief account of the heart-sounds in health embraces, as concisely as possible, the more important of the conclusions deduced from the results of the analysis of the

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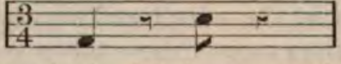
[1] The rhythmical succession of the heart-sounds, musically expressed, may be represented as follows:



In this representation, a bar in triple time corresponds to a revolution of the heart, that is, the period from one beat to another. The crotchet stands for the first sound; the quaver rest for the brief interval between the first and second sound; the quaver for the second sound, and the crotchet rest for the long interval or pause. To show a difference of pitch the first sound is represented by F, and the second sound by C, without, however, attempting to express this difference with musical accuracy.

phenomena obtained by auscultation in the examination of twenty-five persons presumed to be entirely free from disease, the phenomena being carefully noted at the time of the examination. It remains now to notice the modifications of the heart-sounds observed in connection with hypertrophy of the heart. The modifications significant of hypertrophy differ materially from those which pertain to dilatation. The former relate to the present subject. The latter will be noticed in another section in connection with enlargement by dilatation.

Hypertrophy of the left ventricle tends to intensify the element of impulsion of the first or systolic sound. The impulsive movements against the walls of the chest, are proportionate to the hypertrophy of this ventricle. Exceptions to this rule occur when the form of the organ is so changed that the apex fails to come into contact with the thoracic walls. All observers have remarked that in cases of hypertrophy, the first sound is notably dull and prolonged. The dulness and prolongation of this sound, as compared with the second, in health, are due to the element of impulsion. It is, therefore, quite intelligible that when the impulsive movements are increased, the effects on this sound are abnormal dulness and prolongation, as well as exaggerated intensity. Mere exaggeration of this sound is by no means in itself significant of hypertrophy. Increased muscular action of the heart, as in some cases of functional disorder, renders the sound abnormally intense, so that it is sometimes appreciable at a distance from the chest, and painfully perceived by the patient. Both elements of the sound, under these circumstances, are exaggerated. This is also true in cases of pure hypertrophy, *i. e.*, not associated with valvular lesions; but in hypertrophy the element of impulsion is relatively more exaggerated than the valvular element, and hence, the dulness and prolongation are marked, as well as the increased intensity. Modifications affecting the valvular element of the first sound are of importance chiefly in connection with the diagnosis of valvular lesions. The modifications significant of hypertrophy relate more especially to the element of impulsion.

My clinical observations have led me to regard exaggeration of the tricuspid portion of the valvular element of the first sound as evidence, in some cases, of hypertrophy of the right ventricle. To determine the fact of its exaggeration, the valvular element of the first sound is to be compared at the inferior boundary of the heart, near the xiphoid cartilage, with this element at the left border of the heart at or without the left nipple. In health, this element of the first sound is notably more feeble in the former than in the latter situation. If the valvular sound be equally or more marked at the inferior boundary of the heart, provided the mitral valves are sound, it is evidence that hypertrophy of the right ventricle exists, if other signs of cardiac enlargement be at the same time present.

Modifications of the second or diastolic sound, incident to hypertrophy, may affect the aortic and the pulmonary sound separately or combined. The pulmonary and the aortic sound are in relation respectively to the right and left ventricle. The expansion of the semilunar valves succeeding the ventricular systole is due, in a great measure at least, to the systolic contraction of the ventricles. The column of blood propelled from the ventricles dilates the aorta and pulmonary artery, and the recoil due to the elasticity of the coats of these vessels during the ventricular diastole gives rise to the expansion of the valves, and thus occasions the second sound. This is the explanation of the mode in which the expansion of the valves is produced. The force derived from the elasticity of the arteries, it is obvious, must be proportionate to the power of the ventricular systole. The dilatation of the aorta and pulmonary artery is greater the more powerful are the contractions of the ventricles, and the recoil of the arterial coats is stronger the more the vessels have been dilated. Hence, the intensity of the second sound of the heart represents the power of the systolic contractions of the ventricles; and the aortic and the pulmonary sound respectively represent, in this respect, the left and the right ventricle. The two ventricles, as has been seen, may become enlarged by hypertrophy separately, as well as conjointly; and when both are affected the enlargement of one generally predominates over that of the other. It might, therefore, be expected, and clinical observation shows, that an abnormal intensity of the aortic and the pulmonary sound separately, may become a sign of hypertrophy affecting, in the one case, the left, and, in the other case, the right ventricle.

Hypertrophy of the left ventricle gives rise to increased intensity of the aortic second sound, that is, the sound having its maximum of intensity in the second intercostal space on the right side of the sternum, provided this effect be not prevented by attendant circumstances which are of frequent occurrence. Lesions affecting the aortic valves and diminished elasticity of the aorta from disease of its coats; contraction at the mitral orifice, or mitral regurgitation, both lessening the column of blood propelled by the ventricle into the aorta, are circumstances which obviously stand in the way of an abnormal increase of the aortic second sound proportionate to the augmented power of the ventricle. Hypertrophy of the left ventricle is seldom altogether devoid of these circumstances. As a physical sign, therefore, it has little value.

Hypertrophy of the right ventricle, on the other hand, is seldom associated with circumstances preventing its effect on the pulmonary second sound, that is, the sound as heard in the second intercostal space on the left side of the sternum. Lesions of the semilunar valves of the pulmonary artery, and of the tricuspid valves, are of extremely infrequent occurrence. Exaggerated intensity of the pulmonary second sound, therefore, is highly significant of hypertrophy of this ventricle. This effect is especially marked if, in conjunction with increased power of the ventricular systole there exist congestion of the pulmonary vessels involving obstruction to the free passage of blood through the lungs. The resistance which the column of blood propelled into the pulmonary artery then meets with, induces a greater dilatation of this artery during the ventricular systole, and, consequently, a stronger recoil after the systole, thus giving rise to a louder pulmonary second sound. Congestion of the lungs, due to mitral contraction or regurgitation, generally coexists with hypertrophy of the right ventricle, and stands to the latter in the relation of causation. In estimating the amount of exaggerated intensity of the pulmonary second sound, it is to be compared with the aortic second sound in the same intercostal space on the right side of the sternum. In making this comparison, however, it is to be borne in mind that lesions affecting the mitral orifice (contraction, or regurgitation, or both), which are often associated with hypertrophy of the right ventricle, involve diminished intensity of the aortic sound by lessening the amount of blood propelled by the contraction of the left ventricle into the aorta. Under these circumstances, the pulmonary second sound may be more intense than the aortic, although its actual intensity be not augmented. Increased intensity of the pulmonary second sound, occurring in connection with the mitral lesions just named, will be again noticed in treating of these lesions. It is also to be borne in mind that in mere functional excitement of the heart, both the pulmonary and aortic second sound acquire an abnormal intensity. Under these circumstances, the second sound, in both situations, is alike exaggerated. Abnormal increase of the intensity of the sound emanating from either the aorta or pulmonary artery, is more significant of hypertrophy than when the sound from both of these sources is alike augmented. But with respect to the second, as well as the first, sound, abnormal increase of intensity is to be considered as a sign of hypertrophy only when other physical signs of enlargement of the heart are at the same time present.

Enlargement of the heart gives rise to certain abnormal changes as regards the respiratory murmur and vocal resonance within the præcordia, which possess importance as physical signs. In health, the respiratory murmur may, or may not, be perceived within the superficial cardiac region during tranquil breathing; but it is generally heard everywhere within the præcordia when the breathing is forced. In cases of enlargement, however, in which the area of the superficial cardiac region is increased, not only is the murmur in tranquil breathing inappreciable, but it may not be discoverable although the breathing be forced. This is corroborative of the more reliable evidence of enlargement offered by percussion and palpation. The vocal resonance, in health, when more or less marked over the left side of the chest, is either extinct or notably diminished within the præcordial region. The boundaries of the heart may often be as accurately defined by auscultating the voice as by percussion; and, in conjunction with the latter method, the former may be resorted to in determining the augmented space which the heart occupies in cases of enlargement. In females, often, owing to the size of the mammæ, the diminution or extinction of vocal resonance is more available in determining the area of the superficial region than dulness on percussion.

4. *Enlargement of the præcordia and abnormal movements, as determined by inspection.*

In healthy persons, free from spinal curvature, and any deformity of the chest, the præcordial region and the corresponding section on the right side do not present any marked deviation from symmetry. On close comparison with the eye, frequently a slight disparity is perceived, one side projecting a little more than the other. Of the instances, according to my observations, in which this disparity is perceptible, the right and the left side are found to project in an equal proportion. Of twenty-five examinations of different persons in health, with well-formed chests, and no spinal curvature, in seven, no disparity was observable; and in an equal number, viz., in nine, the right and the left side, respectively, were found to be slightly more prominent. Three of these persons were left-handed. In one of these three persons, the right side was more prominent; in another, the left side, and in one there was no disparity.[1]

Abnormal prominence of the præcordial region occurs in certain cases of enlargement of the heart. The prominence is considerable in some cases when the heart becomes enlarged in early life. In a moderate amount, it is not uncommon in cases in which the affection occurs after adult age. Præcordial prominence, due to the accumulation of liquid within the pericardial sac, in cases of pericarditis, may generally be distinguished from that due to enlargement of the heart, by characters determinable by inspection, although the differential signs obtained by other methods of exploration are more strongly marked. The shape of the præcordial projection is not the same in enlargement of the heart as in pericarditis with effusion. In the latter it extends more in a vertical than in a transverse direction. In the former, the arching is wider, and does not extend much, if at all, above the normal situation of the base of the heart. Præcordial prominence due to enlargement, if it exist in a notable degree, denotes both hypertrophy and dilatation, because it is in this species of enlargement that the heart attains to a large size. The projection is very rarely, if ever, so great as in certain cases of chronic pericarditis. The intercostal depressions are not abolished. Bulging of the intercostal spaces, which may result from pericardial effusion, of course, never occurs in cases of enlargement. Widening of the intercostal spaces does not take place to the same extent in cases of the latter as of the former. In enlargement, the apex-beat is generally seen and felt, while in pericarditis it is often suppressed; and if appreciable in the latter affection, it is raised above its normal position, while in the former it is often lowered and carried to the left. Other points of distinction will be noticed in treating of pericarditis. It may be added here that the prominence dependent on enlargement is permanent and unchangeable, while that due to pericardial effusion is sometimes developed under the eyes of the practitioner, and, after variations at different times, may finally disappear and be followed by depression.

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[1] M. Woillez found, of 197 subjects in good health, and without spinal curvature, that in 47 only was the symmetry absolutely perfect. A projection of the left side, in front, either at, or above, or below the nipple, existed in the proportion of 26 per cent. An anterior projection of the right side existed in only two instances. The proportion of instances in which deviation from absolute symmetry existed in my comparatively few examinations agree very nearly with those of M. Woillez. The proportion of instances in which prominence of the left side was noted is larger in my examinations, and the relative number of instances in which prominence of the right side was observed, is still greater.

Movements of impulsion and retraction referable to the heart in cases of enlargement, which have been considered in connection with palpation, are, in general, appreciated by the eye as well as by the touch. Retractive movements may be ascertained by inspection when they are not perceived by palpation. The retraction of the apex-beat is sometimes plainly seen, when an impulse cannot be felt. The alternate movements in different intercostal spaces, which were described as determined by palpation, are best ascertained by inspection. The applicability of this method of exploration to the study of the movements communicated by the heart to the thoracic walls, is to be borne in mind, but it is needless to repeat in this connection the account of these movements, which has been already given.

5. *Increased size of the chest as determined by mensuration.*

The value of mensuration in cases of enlargement of the heart, consists in its giving exactitude to certain of the signs obtained by inspection. It is not essential to obtaining data for diagnosis.

As regards measurements of the healthy chest, with referenc to the præcordia, the following are the conclusions deduced fro twenty-five examinations in which the circumference was measured with graduated inelastic tape, and diametrically by mean of callipers. Equality of the two sides of the chest, and a greate size of the left side, as regards circumference and antero-posterio diameter, do not alone constitute evidence of cardiac or other intra-thoracic disease. This statement holds good within certain limits; in other words, greater size of the left than of the right side beyond half an inch, either by diametrical or circular measurement, points to the existence of disease. Diametrical measurement gives a larger number of instances in which the two sides are equal, than circular measurement, the ratio being six to eleven. The right side was greater in eleven instances as measured by the tape, and in seven as measured by callipers. A greater size of the left side existed in an equal number of instances as determined by the tape and callipers, viz., in five. In all of sixteen cases in which diametrical measurement showed greater size of either the right or left side, the same results had been previously obtained by inspection, with a single exception.

Thus, in confirming and giving greater exactitude to the results of inspection, as respects the size of the chest in cases of cardiac disease, diametrical is to be preferred to circular measurement.

The antero-posterior diameter of the chest at the præcordia is increased in certain cases of enlargement of the heart. In determining that it is due to cardiac disease, abnormal conditions referable to the lungs or pleura, increasing the size of the chest, are to be excluded by the absence of the signs denoting their existence; and the abnormal increase of the diametrical dimensions of the left side is referred to an abnormal condition of the heart, not alone by the exclusion of diseases affecting other intra-thoracic structures, but by concomitant signs of cardiac enlargement. The advantage of mensuration as already stated, is mainly in corroborating the evidence afforded by the eye, and in enabling the physician to determine with greater precision the amount of disparity between the two sides. In recording cases, it is more satisfactory to note the results of a comparison of the two sides in figures than to express them in terms which are somewhat indefinite; such as slight, moderate, great, &c. With reference simply to diagnosis in individual cases, inspection suffices without resorting to measurement.

The diagnosis in cases of enlargement of the heart and of hypertrophy must rest on the physical signs. The symptoms may point to these lesions, and afford corroborative evidence of their existence, but they are not adequate to lead to positive conclusions. So far as concerns enlargement, it is determinable with great ease and precision by means of physical signs in the vast majority of cases. To determine whether hypertrophy or dilatation predominate is more difficult, but in most instances it is practicable with due knowledge and care. As regards these two forms of enlargement, the differential diagnosis will be considered in treating of enlargement by dilatation in an after-part of this chapter. The signs involved in the diagnosis of enlargement and hypertrophy are fewer and more simple than would appear from the space devoted to the subject in this chapter. The subject would here require comparatively brief consideration had it not been requisite, in this connection, to introduce an account of the phenomena obtained by physical exploration in health, as the point of departure for studying the phenomena of disease, not alone with reference to the diagnosis of the affections treated of in this chapter, but of those which are to be subsequently considered. A considerable portion of the present section has been occupied with facts which belong to anatomy and physiology rather than pathology. Having been here introduced, it will only be necessary to allude to these facts hereafter in treating of subjects as preliminary to which they are equally important. For the convenience of the reader, a recapitulation of the physical signs of enlargement and of hypertrophy is given in the summaries which follow.

SUMMARY OF THE PHYSICAL SIGNS OF ENLARGEMENT OF THE HEART.

1. *Percussion.*—The area of the superficial cardiac region extended beyond the range of healthy variation. The degree of dulness within this area greater than in health, and the sense of resistance more marked. The limits of the deep cardiac region, in other words, the boundaries of the heart, generally defined by careful percussion, the dimensions of the space which the heart occupies being thus ascertained with precision, and the form of the organ delineated on the chest. Enlargement of the right or left auricle sometimes determined by the extent of the area of dulness at the base of the heart on the right or left side of the sternum.

2. *Palpation.*—The apex-beat moved to the left of its normal position, and lowered, the extent of variation in either respect

being proportionate to the degree of enlargement, provided extrinsic causes be excluded. The area within which the apex-beat is felt, extended beyond the range of health. Abnormal impulses felt in two, three, or more intercostal spaces; the additional impulses either synchronous or alternating with the apex-beat, in some instances referable to the auricles, although due to the ventricular systole; and, when felt in the epigastrium, due to the action of the right ventricle.

3. *Auscultation.*—The respiratory murmur not appreciable within the superficial cardiac region in tranquil breathing, and sometimes wanting when the breathing is forced; feeble over a larger area within the præcordia than in health. The boundaries of the heart defined by abrupt cessation or notable diminution of vocal resonance, and the augmented space which the organ occupies, in this way determinable in corroboration of the evidence afforded by percussion.

4. *Inspection.*—Abnormal projection of the præcordial region in some cases; the projection considerable if the enlargement take place in early life. The movements of impulsion determined, which are also ascertained by palpation; movements sometimes seen which are not perceptible to the touch, especially movements which commence by depression with the systole of the ventricles. Alternate movements of intercostal spaces often apparent to the eye, which are imperfectly ascertained by palpation.

5. *Mensuration.*—Prominence of the præcordia greater than the corresponding portion of the chest on the right side; in some cases apparent on inspection, but determined with precision by diametrical measurement. Mensuration also employed in determining with accuracy the dimensions of the superficial and deep cardiac regions, the position of the apex-beat relatively to the nipple, the median line, &c.

SUMMARY OF PHYSICAL SIGNS DISTINCTIVE OF ENLARGEMENT BY HYPERTROPHY.

1. *Palpation.*—Abnormal force of the apex-beat, denoting not merely excited action of the heart, but augmented power of the

systole of the left ventricle, the impulsion prolonged, and strong. A strong impulse in the epigastrium in cases of hypertrophy of the right ventricle; the impulsions sometimes communicated to the lower part of the sternum, and extending more or less over the site of the liver. A strong, heaving movement of the præcordia, in distinction from the shock, more or less violent, due merely to augmented functional activity of the ventricles.

2. *Auscultation.*—Increased intensity of the aortic second sound, and especially of the element of impulsion of the first sound, in hypertrophy of the left ventricle, rendering the first sound dull and prolonged, as well as abnormally intense. Exaggerated intensity of the pulmonary second sound, in hypertrophy of the right ventricle, especially if associated with obstruction to the pulmonary circulation. Augmentation of the tricuspid valvular element of the first sound in some cases of hypertrophy of the right ventricle.

TREATMENT OF HYPERTROPHY.

False notions of the pathology of hypertrophy have heretofore led to erroneous principles of treatment. It has been considered an object of treatment to devise the most effective means of diminishing the hypertrophy, and, if this be not practicable, of preventing its progressive increase. For these ends, formerly copious and repeated abstractions of blood were employed, in conjunction with low diet, after the plan of Valsalva and Albertini, Italian physicians. This treatment was found to be pernicious, but, instead of being discarded, the same plan, not carried to the same extent, was recommended by Hope, Bouillaud, and others, and has been generally pursued up to a recent date. A better understanding of the pathological relations of hypertrophy leads to the conclusion that therapeutical measures designed to diminish or prevent it, are likely to do harm in so far as they have efficiency in promoting these ends. Considered in connection with the antecedent morbid conditions which give rise to it, conditions involving impediment to the circulation, hypertrophy, so far from being an evil, is an important provision against the dangers incident to accumulation of blood within the cavities of the heart, and against the evils of dilatation, the latter being much the more serious of the two forms of enlargement. In the great majority of cases, enlargement of the heart is the result of valvular lesions. These lesions often exist for a long time before they give rise to symptoms which lead the patient to suppose that he is affected with disease. When cases first come under the notice of the practitioner, it is evident that the enlargement has been going on for months or even years. The amount of enlargement, when the chest is for the first time examined, shows that it is not of recent production. We have seen that, as regards hypertrophy associated with dilatation, the former, as a rule, takes precedence in time. The hypertrophy, in short, compensates, during a greater or less period, for the disturbance of the circulation caused by the valvular lesions; and so long as the enlargement consists of this compensating increase of muscular structure, and consequently of muscular power, the patient experiences little or no inconvenience, provided nothing occurs, like anæmia, for example, to weaken the force of the heart's action. It is when the hypertrophy has reached the limit of its progress, and dilatation has followed, that serious inconveniences, referable to the heart and circulation, begin to be felt. With this general view of the pathological character of hypertrophy, the indications for treatment may be embraced in three classes, namely: 1. To prevent or limit, as far as practicable, impediment to the circulation which arises from valvular lesions or other conditions, and which thus gives rise to hypertrophy; 2. To obviate, as far as possible, weakness of the heart, and a tendency to dilatation; 3. To quiet undue excitement and irregular action of the heart.

The antecedent pathological conditions giving rise to hypertrophy, namely, valvular lesions, pulmonary emphysema, and Bright's diseases, are not of a nature to admit of removal. The physician, however, can do something towards preventing or limiting the impediment to the circulation, which is the immediate effect of these conditions, and which is the intervening cause of the hypertrophy. This indication is fulfilled by avoiding extrinsic causes which excite unduly the action of the heart, by the employment of measures designed to equalize and tranquillize the circulation, and in some cases by diminishing the contents of the vascular system. Excessive muscular exercise is objectionable, but, as will be seen presently, within certain limits it is not to be prohibited, but enjoined. Excesses in eating and in the use of stimulating drinks are to be avoided.

Mental excitement belongs in the same category. The circulation is equalized by securing, as far as may be, for the different, and especially the remote parts of the body a proper proportion of blood, thus preventing its undue accumulation within the cavities of the heart. For this end, the surface of the body should be guarded against the influence of cold, and revulsive measures, such as warm and stimulating pediluvia, are to be resorted to if the circulation in the extremities be sluggish. Constipation, if it exist, claims appropriate remedies. Bloodletting is permissible only when there exists over-repletion of the general vascular system, the object being, by lessening the mass of blood, to facilitate its circulation. This object should be clearly understood. It is easy to understand that if the vessels be abnormally full of blood, an irremediable impediment to the circulation is likely to occasion greater accumulation in the heart than when the mass of blood to be circulated does not exceed the normal amount. The existence of such a state can alone furnish the indication for bloodletting, and the removal of this state must constitute the limit to which it may with propriety be carried.[1] Carried beyond this limit, the detraction of blood can hardly fail to be pernicious. Injudiciously practised, bloodletting is injurious in proportion as it impoverishes the blood and weakens the muscular power of the heart. The end for which bloodletting is employed may generally be fulfilled by other methods of depletion which involve less risk of doing harm. The use of saline laxatives and diuretics, conjoined with a somewhat restricted diet, and, more especially, with restriction in the quantity of fluid ingesta, will, in most instances, accomplish the object. These means are to be preferred on account of their being free from the evils attending the spoliative effects of bloodletting.

Inconveniences, which hypertrophy alone would not occasion, arise from its association with causes which produce weakness of the heart. All observers have noticed the evils of coexisting anæmia. Impoverishment of the blood renders the heart irritable, easily excited into violent and irregular activity, while its power of action is impaired. Alarming symptoms are sometimes induced under these circumstances, which are so entirely relieved by restoring the blood to its normal condition that patients imagine themselves completely cured. A patient, for example, rendered highly anæmic by lactation, presented dyspnœa, palpitation, and œdema to such an extent that her condition seemed quite hopeless; but after weaning, the use of tonics, &c., she recovered apparently perfect health, so that, except for the physical signs of cardiac disease, the cure would have been considered complete. The combination of anæmia and enlargement of the heart is to be prevented, if possible; and, if it exist, the anæmia, if possible, is to be removed by appropriate measures of medication, diet, and regimen. Irrespective of this condition of the blood, all agencies which tend to weaken unduly the force of the ventricular contractions are contraindicated. In proportion to the weakness of the heart will be the tendency to dilatation rather than to hypertrophy. The latter it would be desirable to promote, were this necessary to prevent the former. So long as hypertrophy predominates, the patient is comparatively safe. The inconveniences and dangers are in proportion as dilatation is superadded to hypertrophy. It is an important object of treatment, therefore, to obviate or retard the tendency to dilatation. With reference to this object, the diet should be nutritious—a substantial, solid diet, adapted to the formation of blood, rich in quality, but not in excess as regards quantity. Muscular exercise within certain limits is to be encouraged rather than repressed. In cases of cardiac disease attended with enlargement, I have been repeatedly struck with the fact that persons engaged in pursuits requiring considerable physical exertions, namely, laborers, mechanics, or active men of business, continue to discharge their duties for a long time without much inconvenience, but become rapidly worse as soon as they discontinue their occupations. I am convinced that a certain amount of exercise is not only allowable, but positively beneficial by promoting the heart's vigor and retarding the progress from predominant hypertrophy to predominant dilatation. It may seem strange to many readers that exercise is recommended in cases of hypertrophy, but, while violent exertions, which excite unduly the action of the heart, are to be avoided, I am satisfied that moderate and sometimes even considerable muscular activity conduces to the welfare of the patient. I can call to mind a number of persons affected with hypertrophy associated with valvular lesions, who, engaged in active occupations, and pursuing no medical treatment, would be amazed were they fully aware of their pathological condition. I cannot but think that were the nature and extent of the disease clearly explained to these persons, and great quietude enjoined, their chances for tolerable health for a considerable period would be materially impaired. Still less encouraging would be the prospect were they subjected to a course of diet and medication tending to impoverish the blood, reduce the vital forces, and weaken the power of the heart. I cannot avoid the conviction that I have witnessed injury inflicted by this course of management in not a few cases.

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[1] It is assumed that an abnormal augmentation of the mass of blood may exist, and that when the mass of blood is diminished by bloodletting or other means, the vessels are not immediately refilled. The assumption of these points, in opposition to the speculative views of some, is believed by the author to be in accordance with clinical observation.

In some cases of hypertrophy, the heart is unduly excited, and irregular action takes place, even when extrinsic causes are, as much as possible, avoided. In other words, functional disorder, or palpitation, may be superadded to the organic affections. This is not only a source of inconvenience, but there is reason to believe that the effect is unfavorable as regards the permanent condition of the heart. To quiet undue excitement and irregular action of the heart, is therefore in these cases, an object of treatment. Certain remedies may be employed with advantage for this object. Digitalis is found frequently to produce a sedative effect without lessening the power of the heart's action. Under its judicious use the ventricular contractions may become less frequent, more regular, and apparently more complete. Aconite is a useful remedy for the same object. The veratrum viride, if used for this object, should be employed with great circumspection, in order to avoid its depressing effect. Belladonna is useful. A belladonna plaster worn over the præcordia has some effect in tranquillizing the action of the heart. The sedative effect of the hydrocyanic acid is sometimes useful.

In these remarks on the treatment of hypertrophy, I have not discussed the feasibility of diminishing the abnormal growth of the muscular walls of the heart, a subject concerning which different writers have held opposite opinions. The views of the pathological character of hypertrophy which have been presented, divest this subject of the practical importance which has heretofore been attached to it.

ENLARGEMENT BY DILATATION.

Under this head are embraced, in addition to the rare cases of pure or simple dilatation, that is, those in which the capacity of the cavities is increased and the walls attenuated, all cases in which dilatation coexists with, but predominates over, hypertrophy. Of the two kinds of enlargement, this is by far the most frequently found after death when organic disease of the heart proves fatal. In the cases in which the heart attains to a very large size, dilatation almost invariably preponderates. The cases in which the organ, from its immense bulk, resembles a bullock's heart (*cor bovinum*), are those in which there exists great hypertrophy, with still greater dilatation. The degree of dilatation varies greatly in different cases, and the hypertrophy combined with it is also variable. The preponderance of dilatation, when the heart is examined after death, is generally sufficiently evident on inspection. The abnormal increase in the dimensions of the organ exceeds that of the weight. The ventricular walls collapse, and the organ, resting on its posterior surface, is flattened, instead of preserving a globular form, as when hypertrophy predominates. The greater increase in width than in length, is marked in proportion to the preponderance of dilatation. Owing to this, the organ becomes wedge-shaped, and sometimes presents nearly a square form.

The mechanism of dilatation is quite different from that of hypertrophy. In the production of the latter, the process is vital, whereas in the former, it is mechanical. Hypertrophy is a consequence of over-nutrition; dilatation is the result of the yielding of the walls of the heart to a distending force, the condition, however, which stands immediately in a causative relation to both is the same, viz., undue accumulation of blood within the cavities of the heart; hence it is that both take place either conjointly or in succession, so that hypertrophy and dilatation are very often associated. Dilatation, thus, not less than hypertrophy, depends on antecedent affections which occasion impediment to the circulation through the vessels or the orifices of the heart, leading to over-accumulation of blood within the cavities. These antecedent affections are the same as in cases of predominant hypertrophy; and the several portions of the heart become affected singly and in succession, as in the latter form of enlargement. It is not necessary, therefore, in this connection, to consider the dilatation of these portions, respectively, in relation to the particular lesions of the valves and orifices and vessels on which dilatation and hypertrophy alike depend. Moreover, both dilatation and hypertrophy of the different portions of the heart will be referred to hereafter in treating of valvular lesions. It will suffice to inquire into the circumstances which determine the occurrence of dilatation in the place of, or, as is generally the case, in addition to, hypertrophy.

The first effect of an undue accumulation of blood in the cavities of the heart, continued for a sufficient period, is increased power of muscular action and consequent hypertrophy in the great majority of cases. The hypertrophy is more or less progressive, but it has its limit. The abnormal growth of the muscular tissue ceases after it has progressed to a certain extent. But the morbid conditions inducing over-repletion of the cavities still remain, impeding more and more the circulation. The compensating increase of the muscular tissue no longer taking place, the walls of the cavities yield to the mechanical force of distension, and the progressive enlargement from this time onward is due to dilatation. The limit of hypertrophic enlargement varies in different persons. If the hypertrophy progress until the muscular walls attain to a great thickness, and life continue for a long period afterward, dilatation finally predominates, and the result is an enormous enlargement of the heart, a cor bovinum. But dilatation may commence after moderate or slight hypertrophy has taken place; in other words, the hypertrophy ceases after a smaller amount of muscular growth, and dilatation commences. Dilatation may even commence without any previous hypertrophy, and the result is, then, enlargement with attenuated walls, or simple dilatation, a rare variety of enlargement of the heart. The occurrence of dilatation is determined by the state of the muscular walls. Functional debility of the organ, and still more, changes in the muscular fibres prevent that vigorous activity which induces abnormal growth, and yielding of the walls takes place early in proportion as the vital power of resistance is impaired. Anæmia, the feebleness consequent on pericarditis and adherent pericardium, fatty degeneration, softening, and any changes which compromise the muscular power of the organ, tend to abridge hypertrophy and favor dilatation. The latter will therefore predominate in proportion as the condition of the walls is such that they early and readily yield to the distension caused by the accumulation of blood within the cavities. After this brief consideration of the circumstances determining the occurrence of dilatation, in addition to the incidental remarks already made under the head of enlargement by hypertrophy, the reader will be able to trace the relations of dilatation affecting the different cavities of the heart to lesions of the mitral and the aortic orifice, involving either obstruction or regurgitation, or both, and to obstructions affecting the pulmonary and systemic arterial systems at situations more or less remote from the heart, without recapitulation of the account already given in connection with hypertrophy. The inquiry arises, Does not the heart in some instances become dilated in consequence of inherent weakness, no antecedent affections existing to occasion impediment to the circulation? It is probable that this sometimes occurs as an effect of fatty degeneration, pericardial adhesions, atrophy or softening of the muscular fibres, &c. Examples are found of dilatation associated with these structural changes, and without other obvious sources of impediment to the circulation. These changes may be subsequent to dilatation, but it is reasonable to suppose that in some instances they precede and give rise to it. Clinical observation, however, furnishes no evidence that functional weakness alone leads to dilatation, irrespective of structural changes of the walls of the heart, or lesions of some kind which occasion impediment to the circulation. Dr. W. T. Gairdner accounts for dilatation of the heart, in cases of pulmonary emphysema, in the same way that he accounts for emphysema, namely, the cavities of the heart are dilated by the force of inspiration, as are the unobstructed air-cells in consequence of collapse of more or less of the pulmonary lobules. This author accounts in this way for, not only dilatation, but hypertrophy, the expansion of the thorax tending constantly to overload the heart, and thus occasioning increased muscular force. Without adopting this explanation, it must be admitted that Dr. Gairdner bases his explanation on facts which have considerable weight. "Of 24 cases of enlargement without valvular lesions, in 21 cases there were manifest and extensive old atrophic lesions of the lungs, with or without accompanying emphysema, which is recorded as having existed in 17 of the cases." Dr. Gairdner, also shows by an analysis of fatal cases, that enlargement of the heart occurs oftener in emphysematous cases than in mixed cases, the proportion being as 15 to 23 per cent.; and that, of the cases occurring with emphysema, valvular lesions are present in a less proportion than in the mixed cases, the whole number of cases analyzed being 84. To show that contraction of the capillaries of the lungs from any cause, or obstructed circulation through these organs, will not give rise to enlargement of the heart, he analyzes 18 cases in which effusion into the pleuræ or peritoneum, or other causes, occasioned compression of the lungs for a considerable time, there being no valvular lesions. In only four of these cases did enlargement exist, and in one case its existence was doubtful. On these data he bases the conclusion, that, while diseases of the lungs which merely obstruct or obliterate the circulation in the capillaries have no well-marked tendency to be associated with enlargement of the heart, those which produce atrophy of the pulmonary tissue and secondarily emphysema have an obvious influence on the heart, and frequently cause its enlargement.

SYMPTOMS AND PATHOLOGICAL EFFECTS OF DILATATION.

The symptoms due to dilatation, like those of hypertrophy, are generally involved with those incident to valvular or other concomitant lesions. In proportion to the amount of dilatation, the muscular power of the heart is impaired. The symptoms distinctive of dilatation proceed from feebleness and incompleteness of the heart's action. The action of the heart is often irregular, as represented by irregularity of the pulse and of the apex-beats. Both are abnormally feeble. The pulse may be unequal as well as irregular. These symptoms are in relation to dilatation of the left ventricle. The patient experiences more or less uneasiness and undefinable distress, referable to the præcordia, but he is not conscious of that powerful action of the heart which characterizes hypertrophy. The extremities and surface of the body are cool. Lividity may be apparent on the prolabia, the tongue, face, and extremities. The veins, especially those of the neck, may be distended. These symptoms are more or less marked in proportion as the dilatation affects the right ventricle. Dyspnœa will be prominent in proportion as the right ventricle is the seat of dilatation. The recumbent posture, with the head low, may be insupportable, and in an advanced stage, the suffering from defective hæmatosis may amount to orthopnœa. Occurring in paroxysms, this difficulty of respiration has been called cardiac asthma. Exercise, and mental excitement augment the symptoms, particularly the dyspnœa. More or less cough is usually present, with serous and sometimes sanguinolent expectoration. The abdominal viscera, as well as the lungs, are in a state of passive congestion. Owing to this state, the liver may become more or less enlarged, and may be found to augment rapidly in size when, from any cause, the circulation is temporarily embarrassed in an unusual degree, resuming its former dimensions when the paroxysm ends and the heart recovers its habitual strength.[1] The digestive functions are weakened, but nutrition may be sufficiently active; patients do not always emaciate. The urine is not abundant, and may be found slightly albuminous, which is due to renal congestion, and is not necessarily indicative of structural disease of the kidneys. Renal disease is, however, associated, in a certain proportion of cases, with dilatation as with hypertrophy. Finally, œdema occurs, first, manifested in the lower extremities, thence extending over the body, and effusion into the serous cavities succeeds, constituting general dropsy.

This is an enumeration of the more important of the symptoms belonging to cases of enlargement in which dilatation predominates, but it is to be borne in mind that, in general, valvular or other lesions coexist, which, after inducing more or less hypertrophy, have at length led to dilatation ; and, under these circumstances, it is difficult to say to what extent the symptoms distinctive of this stage of the disease may not be due to the causes of the dilatation, in other words, to the concomitant lesions. But it is certain that much, if not chief importance is to be attached to the dilatation in the production of the symptomatic phenomena which have been mentioned.

The pathological effects of dilatation are in a great measure embraced in the foregoing account of the symptoms. The dilatation is the result of weakness of the cardiac walls, together with an accumulation of blood within the cavities; and, on the other hand, it is the cause of further diminution of the power of the heart's action, and consequent over-repletion. It has, therefore, an intrinsic tendency to increase. The evils incident to enlargement are mostly referable to dilatation. Little or no inconvenience is felt so long as the heart is hypertrophied, and the capacity of its cavities not increased. But in proportion as the latter takes place, the quantity of blood to be propelled from the cavities is greater, and the ability of the muscular walls to contract sufficiently for its propulsion is lessened; hence, inadequacy of the motive power of the central organ to carry on the circulation. This inadequacy increases in more than an arithmetical ratio as the dilatation progresses. The immediate effect on the vascular system is passive congestion, arising not alone from the defective propelling power of the heart, but from the obstacle presented to the return of blood to this organ by the accumulation within its cavities. The ulterior effects dependent on congestion are, embarrassment of the functions of the important organs of the body, serous transudation or dropsy, and, occasionally, hemorrhage. An occasional effect of great dilatation conjoined with extreme feebleness of the heart's action, is the formation of coagula within the cavities of the heart. There is reason to believe that in some instances in which the accumulation is excessive, and the contraction of the walls extremely feeble, the blood coagulates during life, and proves the immediate cause of a fatal termination. An unusual accumulation of blood, from any cause, in either the right or the left ventricle, when it is much weakened by dilatation, may occasion paralysis of the walls by distension, and thus produce sudden death.

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[1] Stokes on the Heart and Aorta.

Some of the French writers apply the term asystole to such a degree of incompetency of the ventricular contractions, as to give rise to the foregoing symptoms and pathological effects. This term asystole, denoting, not, of course, absence of the systolic movements, but a notable diminution of their power, was introduced by Beau, and is applied to incompetency of the systole of the ventricles, incident, not only to dilatation, but to other cardiac lesions, and even to purely functional weakness existing in a marked degree.

PHYSICAL SIGNS DISTINCTIVE OF ENLARGEMENT BY DILATATION.

The physical signs of enlargement of the heart have been already fully considered. The signs distinctive of dilatation are now to be noticed. The several methods of exploration which furnish evidence of enlargement, contribute signs pointing to dilatation in distinction from hypertrophy. The evidence obtained from percussion relate to the form of the area of deep dulness. If the boundaries of the heart be delineated on the chest by means of percussion, the transverse dimension of the area exceeds the vertical in proportion as dilatation predominates over hypertrophy. This corresponds to the difference as regards the form of the heart, which has been stated. The outline which the heart presents is wedge-shaped or nearly square if the dilatation be excessive. Palpation furnishes negative characters more readily available and striking. The powerful apex-beat of hypertrophy is wanting; also the elevation of the ribs and the heaving of the præcordia. The impulse of the apex is feeble, and may be suppressed. The movements of the organ, owing to the extended space in which it is in contact with the thoracic walls, are sometimes obscurely felt, and oftener visible in two, three, four, or even more intercostal spaces, which together present an appearance of fluctuation, or, as called by Walshe, quasi undulation. In some cases in which the thoracic walls are thin, and the intercostal spaces wide, the heart seems to be almost exposed to the vision and touch. Auscultation furnishes certain distinctive points pertaining to the heart-sounds. Both sounds are feeble in comparison with their augmented intensity in cases of hypertrophy, but the first sound is disproportionately weakened. The first sound is also altered in character; it becomes short and valvular, resembling in these respects the second sound. The latter alterations, although distinctive of dilatation as contrasted with hypertrophy, are not peculiar to the former, and their true explanation has not been understood. They are due to the impairment or absence of the element of impulsion in the first sound. This element is deficient or wanting whenever the left ventricle lacks the muscular power necessary for its production. In hypertrophy this element is intensified, owing to the increased force of the ventricular contractions; and in dilatation it is feeble or absent, owing to the feebleness which, at the same time, renders the apex-beat weak or inappreciable. But this element is also impaired or absent when, from other causes than dilatation, the muscular power of the heart is weakened. The intensity of the first sound is diminished disproportionately to that of the second sound, and it is also short and valvular like the second sound, in cases of fatty degeneration, and of softening in typhus fever, and of pericarditis with effusion. The valvular element predominates, or is alone present, in consequence of the feebleness or absence of the element of impulsion. But the intensity of the valvular element is also more or less diminished, in the first place, in consequence of the weakness of the ventricular contractions, and in the second place, because at the time when the ventricular contractions take place the quantity of blood within the ventricles is large, causing closure of the auriculo-ventricular valves.

An adventitious sound or murmur accompanies the first or systolic sound in some instances of dilatation not complicated with valvular lesions. As a rule, a murmur is not present unless valvular lesions coexist, or the blood have undergone those changes which occasion a murmur without any organic affection of the heart. This point will be noticed in treating of murmurs in connection with valvular lesions. Inspection shows in certain cases the quasi-undulatory movements within the præcordia which have been mentioned as determinable by palpation. They are better perceived by the eye than by the touch. Inspection and mensuration may show an abnormal prominence of the præcordia. In the rare cases of dilatation with attenuated walls, it may be true that enlargement of the præcordia never occurs. This is not true, however, of all the cases in which dilatation predominates over hypertrophy. Without discussing the question whether enlargement by dilatation, as well as by hypertrophy, may not give rise to præcordial projection, this result may be produced by the hypertrophy before the supervention of dilatation which subsequently becomes predominant. Absence of præcordial prominence does not then belong among the negative signs of enlargement by dilatation.

In the diagnosis of enlargement by dilatation, assuming the fact of enlargement to have been ascertained, symptoms (as distinguished from signs) have considerable weight. Passive congestions, lividity, feeble pulse and dropsical effusion, in fact, constitute evidence almost, if not quite, conclusive. The obstruction due to the valvular lesions which are so generally associated with enlargement, it is true, contributes toward the production of these symptoms; but, as will be seen when valvular lesions are considered, the obstruction due to these rarely, if ever, gives rise to the effects just mentioned until dilatation of the cavities of the heart has taken place. With the aid of the physical signs, the discrimination between predominant dilatation and predominant hypertrophy may generally be made with confidence. The differential diagnosis is of importance with reference to prognosis and treatment. The prospect of life and tolerable health is less in proportion as dilatation predominates, and the management involves attention to incidental events which do not occur as long as hypertrophy predominates. For the convenience of comparison with the physical signs distinctive of hypertrophy (see page 73), the signs distinctive of dilatation are embraced in the following summary.

SUMMARY OF THE PHYSICAL SIGNS DISTINCTIVE OF ENLARGEMENT BY DILATATION.

1. *Percussion.*—The transverse dimensions of the space occupied by the heart greatly exceeding the vertical, the form of this space corresponding to the wedge-like or square form of the organ when the dilatation is excessive.

2. *Palpation.*—The apex-beat devoid of abnormal force and in some instances suppressed. Absence of heaving movement of the ribs and præcordia.

3. *Auscultation.*—The element of impulsion of the first sound deficient or absent, and the sound short and valvular, in these respects resembling the second sound.

TREATMENT OF DILATATION.

With certain qualifications, the indications for the treatment of dilatation are the same as in cases of predominant hypertrophy. The impediment to the circulation dependent on the lesions which coexist in the great majority of cases cannot be removed, but the effects may be mitigated by avoiding extrinsic causes which excite unduly the action of the heart. Limiting the attention to the diminution of the mass of blood, it might seem that bloodletting would fulfil an important indication. But it is to be considered that bloodletting impoverishes the blood, and the secondary consequences are weakness and irritability of the muscular structure of the heart. These consequences are hurtful to an extent greatly overbalancing the advantage of temporarily diminishing the quantity of blood. No advantage to be derived from this measure can compensate for the evils of anæmia. Bearing in mind the immediate effects of bloodletting on the composition of the blood, and the secondary effects, due to impoverished blood, on the muscular structure, the cases in which it is called for seldom, if ever, occur.' These remarks will, measurably, but not nearly to the same extent, apply to other methods of depletion, namely, saline purgatives and diuretics. Excessive muscular exercise, mental excitement, and other extrinsic causes exciting unduly the action of the heart, are to be avoided. Warmth of the external surface, and revulsive measures to attract blood to the extremities, are indicated oftener and more strongly in cases of dilatation than in cases of hypertrophy.

The measures which in hypertrophy are pursued in order to prevent dilatation, are not less indicated when the latter exists. The great end in the management is to increase the muscular power of the heart. For this end, the diet should be as highly nutritious as possible, and the quantity of liquid ingesta as small as is compatible with comfort. In the arrangement of diet, the state of the digestive organs is to be consulted. Imperfect or labored digestion involves excited action of the heart, and is to be carefully avoided. When indigestion exists, palliative remedies are to be prescribed ; and remedies to improve the digestive function, namely, tonics and the judicious use of stimulants, constitute an important part of the treatment. Preparations of iron are especially indicated if there be anæmia. Constipation is to be prevented. Exercise, within certain limits, is to be enjoined. The injury arising from excessive muscular exertion has been referred to; but an extreme of quietude is not less hurtful. How is the judicious mean to be determined? The experience of the patient must be the guide. An amount or a kind of exercise which excites unduly the action of the heart or occasions dyspnœa is to be abstained from; but exercise short of these effects will be useful. Patients who follow avocations which involve manual labor will, in general, do better to pursue their callings, observing the precaution just mentioned, than to relinquish all occupation. The necessity for an undue amount of labor in order to obtain a livelihood is a calamity for persons affected with cardiac disease; but a condition in life in which there is no other motive for exertion than the attainment of health is sometimes equally calamitous. Patients of the latter class should be encouraged to engage in sports which afford the requisite exercise, and, at the same time, interest the mind, such as shooting, fishing, and travelling. An advantage of no small account, incidental to pursuits which involve both exercise and mental occupation, accrues from the diversion of mind and cheerfulness which they promote. Depression and gloomy forebodings are to be obviated as far as possible, and with a view to this, as much encouragement should be given as the nature of the case will permit. In a large proportion of the cases which the physician meets with in practice, he may conscientiously encourage hopes, not of cure, but of tolerable health for an indefinite period. The common notion that disease of heart generally ends in sudden death may be removed by positive assurances of its falsity.

Remedies to allay undue excitement and irregularity of the heart's action are indicated in cases of dilatation, as well as in cases of hypertrophy. As a remedy with reference to irregularity of the heart's action incident to dilatation, digitalis often manifests a truly remarkable efficacy. Of the different preparations the tincture is to be preferred on account of its being more reliable as regards strength. Digitaline, however, has still more this advantage, and is, perhaps, entitled to preference. With respect to this remedy in cases of enlargement both by hypertrophy and by dilatation, there has been much discussion, and there is a singular discrepancy of opinion among different clinical observers. Some consider it a remedy which weakens the heart, and therefore not indicated in dilatation. It has even been supposed that sudden death may be sometimes attributable to the use of this remedy. On the other hand, others, regarding it as a cardiac tonic or stimulant, consider that the weakness of the heart incident to dilatation is an indication for its use, and that it is contraindicated by hypertrophy. Without discussing these different opinions, it may be assumed that, given in small or moderate doses, for example, from ten to thirty drops of the tincture, it cannot, under any circumstances, have much potency to harm, and it cannot prove a dangerous remedy. That it renders the action of the heart slower and more regular is undeniable. With reference to these effects, Bouillaud calls it "the opium of the heart." That it produces these effects without weakening the organ may be assumed, and clinical observation appears to show that, under its use, the heart, already weakened by dilatation, acts with increased strength. With these views, it is a remedy useful in cases both of hypertrophy and of dilatation. Aconite is also useful, and, perhaps, also, the veratrum viride; but these and other cardiac sedatives should not be pushed beyond the effect of tranquillizing the action of the heart.

The paroxysms of dyspnœa or orthopnœa, sometimes the source of great distress in cases of dilatation, are to be palliated by antispasmodic remedies and revulsive applications. Of the former, the ethers, and of the latter, sinapisms, dry cupping, and stimulating pediluvia, are the most efficient.

The treatment of dropsy dependent on cardiac disease is deferred until after the consideration of valvular lesions.

CHAPTER II.

LESIONS, EXCLUSIVE OF ENLARGEMENT, AFFECTING THE WALLS OF THE HEART.

Atrophy of the heart—Fatty growth and degeneration—Symptoms and pathological effects of fatty growth and degeneration—Physical signs and diagnosis of fatty growth and degeneration—Treatment of fatty growth and degeneration—Softening of the heart in typhus and typhoid fever and other affections—Treatment of softening of the heart—Induration of the heart—Cardiac aneurism and aneurism of the coronary artery—Rupture of the heart—Carcinoma, tuberculosis, extravasation of blood and cysts.

Exclusive of enlargement, the heart is liable to various lesions affecting the walls of the organ. Atrophy is one of these; fatty growth and degeneration constitute others; other lesions are, softening and induration, and in this category may be included aneurism of the heart and rupture. This chapter will be devoted to the consideration of these different organic affections, taken up in the order in which they have just been mentioned.

ATROPHY OF THE HEART.

The muscular substance of the heart is sometimes diminished, the cavities not being enlarged, but, on the contrary, their capacity lessened. The organ is reduced in size below the normal limits. In the adult subject it may resemble in bulk the heart of a child. The weight corresponds to the diminution in size. This reduction in size and weight does not involve necessarily any change in the appearance of the organ in other respects, the only deviation from the normal condition being the diminution in volume and in the thickness of the ventricular walls.

This is undoubtedly to be considered as an organic affection of the heart, but it very rarely, if ever, occurs except in harmony, so to speak, with other morbid conditions, and under circumstances in which it neither occasions unpleasant consequences, nor claims attention in a therapeutical point of view. It is incidental to chronic diseases of long duration, characterized by gradual, progressive emaciation. It is observed in some cases of pulmonary tuberculosis, and more especially in cases of carcinoma. It follows, in some instances, pericardial adhesions and calcification of the coronary arteries. It sometimes exists in connection with a superabundance of fat on the exterior of the heart, and may be due, in these instances, as in cases of pericardial adhesions, to mechanical pressure of the organ continued for a long period. The conditions giving rise to it are diminution of the mass of blood, and of its nutritive materials—conditions involving diminished exertion of the muscular power of the organ. The heart wastes like other muscles when badly nourished and insufficiently exercised. But, under the circumstances, that is, in view of coexisting tuberculosis or carcinoma, or some other affection, which, like these, terminates fatally after slowly progressive emaciation, the atrophy, so far from being an evil, may perhaps belong among the conservative provisions of which the pathological history of even the most fatal forms of disease furnishes illustrations.

The symptoms of atrophy of the heart in these cases, it is sufficiently clear, must be those which denote feebleness of the circulation; but inasmuch as an enfeebled circulation due to other morbid conditions, precedes and gives rise to the atrophy, it must be difficult to decide to what extent the symptoms are dependent on the latter. Nor are the symptoms denoting feebleness of the circulation distinctive of this particular lesion of the heart. They are incident alike to dilatation, fatty degeneration, softening, &c. The physical signs are more distinctive, and, in fact, suffice for the diagnosis. The boundaries of the superficial and deep cardiac regions are within the extreme limits of health; the apex-beat is indistinct or wanting, and the heart-sounds are abnormally feeble, and may be inappreciable.

This form of atrophy may be congenital. It is observed in connection with the arrest of development of other organs, more especially the genital organs, and it is oftener observed in females than in males.

To atrophy with diminished volume and weight of the heart, may be added degenerative disease of the muscular structure of the organ. Fatty degeneration is the disease oftenest associated. The presence of dark or brown granules in the place of the muscular substance constitutes a variety which has been called pigmentary atrophy. With the latter fatty degeneration is generally combined.[1] These degenerative lesions diminish the muscular power of the heart out of proportion to the diminution in volume and weight of the organ. The smallest heart which has fallen under my observation, weighed four and a half ounces; the walls of the left ventricle were not over a quarter of an inch in thickness, and the cavities were not enlarged. The muscular walls of the ventricle were flabby and of a fawn color. The structure was not examined microscopically, but it can scarcely be doubted that fatty degeneration existed. Fatty liver in a marked degree coexisted. The patient, in this case, was a female, who was admitted into hospital with pleurisy, accompanied with large effusion, on the left side. Thoracentesis was twice resorted to with temporary relief. Death took place by asthenia. Feebleness of the pulse was a notable feature; it was scarcely appreciable during the latter weeks of life.

A specimen of brown or pigment atrophy came under my observation in a hospital case of death from gastric ulcer. The heart weighed 7 ounces. On section the divided surfaces had a slate or bronzed color. The liver in this case was notably small, weighing only 29 ounces.

Atrophy with increased size of the cavities, the volume of the heart being greater, and the weight less, than in health, has been already referred to in treating of dilatation. It does not claim distinct consideration as a variety either of dilatation or atrophy. I presented a specimen at a meeting of the New York Pathological Society, March 12, 1868, in which the muscular substance of the right ventricle was notably diminished, and over a space of about the size of half a dollar, the muscular structure had nearly disappeared, a few fibres only being seen when the ventricular side was held between the eyes and a strong light. Fibroid tissue had taken the place of the muscular structure. The patient, in this case, entered hospital with pleuro-pneumonia, and fell dead in walking from the water-closet to his bed. The right ventricle was greatly dilated and filled with dark, loose coagula. There were no valvular lesions. The sudden death was attributable to paralysis of the heart from distension caused by an accumulation of blood in the right cavities.

Atrophy of the heart may exist in a marked degree, although the weight, as well as volume, of the organ is increased. The accumulation of fat may be sufficient to cause enlargement, as regards both volume and weight, while the muscular substance is diminished. Carcinoma and other morbid products, in the same way, lead to atrophy, and also to enlargement. These changes, however, fall under other heads than atrophy.

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[1] Raynaud, op. cit.

Atrophy, denoted by diminished volume and weight, without degenerative disease, does not call for treatment, as already stated. In these cases, as a rule, the atrophy affects about equally all portions of the heart. The treatment when associated with degenerative lesions, either with, or without, increase of weight or volume, will be considered in other subdivisions of this chapter.

FATTY GROWTH AND DEGENERATION.

With the undue accumulation of fat are connected lesions quite different in character and importance, according to the difference of situation in which the fat accumulates. More or less fat is generally present in health on the outer surface of the heart after early infancy, especially on the right ventricle, at and near the base of the organ. It is most abundant between the ventricle and auricle, and around the coronary vessels. It accumulates to an abnormal extent in some cases. A moderate amount of over-accumulation is frequently met with in post-mortem examinations, when there had been during life no symptoms of disease of the heart. If the quantity do not considerably exceed the normal average, although it must in some measure embarrass the movements of the organ, it does not occasion any appreciable inconvenience. When the accumulation is excessive, however, from its weight it leads to enfeebled muscular action and consequent weakness of the circulation. It may also favor dilatation if, from other causes, the blood accumulate unduly within the cavities of the heart. Without these concurrent causes, it may induce atrophy with diminished size of the muscular portion of the heart. This variety of fatty heart occurs after the middle period of life, in persons who present evidence of an "adipose diathesis,"[1] viz., accumulation of fat in different organs and beneath the integument, constituting corpulency. Not infrequently, however, it occurs in persons who are not corpulent.[1] The heart is sometimes completely encased in a thick layer of adipose substance, which alters, in a marked degree, the external appearance and form of the organ. The volume of the heart is often increased, not alone by the fatty deposit, but by more or less dilatation. Beneath the fatty layer the muscular substance may not present any structural change. It is, however, generally unusually pale, and the texture softened.

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[1] This term is borrowed from my friend, Prof. Gross. Elements of Pathological Anatomy, third edition, 1857. Dr. Bellingham also makes use of the term "fatty diathesis." Treatise on Heart, part ii, 1857.

An accumulation of fat between the muscular fibres is followed by more serious consequences than when the deposit is limited to the surface of the organ. The pressure upon the fibres causes functional weakness, and is more likely to induce atrophy. The power of the heart in propelling the currents of blood and in resisting the force of distension from accumulation within the cavities is proportionately lessened. Hence, feebleness of the circulation and proneness to dilatation in proportion to the amount of deposit in this situation. The deposit in this situation may be in the form of adipose vesicles and infiltrated oily matter. Generally when fat accumulates between the muscular fibres there is more or less over-accumulation upon the surface of the heart; but exceptionally it accumulates in the former and not in the latter situation.

Another variety, much more serious, and differing essentially in character, is called fatty degeneration. The fat, in the form of oil-drops or granules, replaces the muscular substance and constitutes a form of fatty atrophy. This variety may be associated with the preceding varieties of fatty heart, but it occurs independently of the latter. It affects more especially the left ventricle, while the varieties consisting of abnormal growth of the adipose vesicles are most abundant on and within the right ventricle. It may be pretty uniformly diffused over the left ventricle or over the whole heart, but it is oftener confined to circumscribed patches or strips. The portions affected assume a yellowish or fawn color, which is somewhat characteristic, and if the heart be affected in disseminated patches it presents a mottled aspect. Examined with the microscope, the striæ or transverse markings of the fibres are indistinct or wanting, and in place of the proper constituents of the muscular fibre there are oil-globules and granules in more or less abundance according to the amount of degeneration.

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[1] Of 49 cases analyzed by Dr. T. K. Chambers (Decennium Pathologicum), it was associated with general corpulence in 20, and occurred in persons not corpulent in 29. Vide Bellingham, op. cit., part ii.

The fatty degeneration may be limited to, or more marked in, either the outer or the inner layer of muscular fibres composing the ventricular walls. It may extend to the papillary muscles and the interventricular septum. The portions which have undergone fatty degeneration are softened, and, as will be seen presently, when rupture of the heart takes place it is owing generally to this structural change having occurred. If the degenerative change have extended over a considerable portion of the heart, the organ is flabby, the walls collapse, and the incised surfaces give to the touch an unctuous sensation. The softening is shown by the little force required to penetrate the muscular substance with the finger. It is evident that in proportion to the degree and extent of this structural change the muscular power of the heart must be impaired. In so far as fat replaces the muscular substance, the heart is incapacitated to propel the blood with adequate force, and it more readily yields to distension from the accumulation of blood in the cavities. In some specimens the larger part of the muscular substance is found to have disappeared, the fibres preserving their outline, but consisting of fat in place of their proper anatomical elements.

The distinct pathological character of fatty degeneration, as compared with fatty growth upon the heart and between the muscular fibres, is a point of importance. The term *degeneration* implies a conversion of the muscular substance into fat. That the mechanism of fatty degeneration does involve this transformation is the view entertained by some distinguished pathologists.[1] The muscular substance, according to this view, undergoes a metamorphosis, the elements recombining to form fatty matter, as muscular tissue after death is supposed to be converted into adipocere. If this view be correct, it is not strictly accurate to call the fatty matter a deposit; it is not, at all events, deposited primarily as fat, but as the substance of the muscle. Nor is the change due to perverted nutrition: it is due to a chemical, not a vital process. Another explanation attributes the change to a process of replacement rather than conversion, the fat being an abnormal deposit, which takes the place of the muscular substance. The change, agreeably to this explanation, does not consist, properly speaking, in a degeneration of structure, but in the substitution of one anatomical element for another, and it has been proposed to employ, as a more accurate mode of expression, the term *substitution* instead of degeneration.[1] They who adopt the latter view regard atrophy of the muscular tissue as the first step in the local pathological process. The anatomical elements disappear by absorption, and fat is deposited in their place. It is, perhaps as reasonable to suppose that the primary change is the fatty deposit, the removal of the anatomical elements of the muscular tissue taking place subsequently, the atrophy thus being not a prior, but a consecutive condition.

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[1] For the evidence to be adduced in support of this doctrine, the reader is referred to an article by Dr. Richard Quain (in Medico-Chirurgical Transactions, vol. xxxiii) on Fatty Disease of the Heart.

On what antecedent morbid conditions are the different forms of fatty disease of heart dependent? Morbid growth or hypertrophy of the adipose tissue, as already stated, is often associated with that tendency to superabundance of fat which constitutes obesity. This tendency is directed toward the heart, after middle life, in persons of indolent and luxurious habits, and in those who are addicted to the use of alcoholic beverages. Active exercise and a well-regulated diet serve to protect the heart against accumulation of fat, even when the adipose diathesis is marked. Fatty degeneration occurs independently of this diathesis. It is a question whether it involves a prior alteration of the blood, or is to be regarded as an effect of conditions purely local. The latter view is favored by the doctrine that muscular atrophy precedes the fatty deposit; and, on the other hand, if the deposit take precedence, a predisposing condition of the blood is reasonably inferred. The latter supposition is sustained by the fact that, although this variety of fatty heart occurs in persons who are not corpulent, it is usually found in association with fatty degeneration in other parts, especially the liver, spleen, and the arterial coats. Prof. R. W. Smith, of Dublin, has reported cases in which free oil was collected, after death, in considerable quantity, from the blood contained in the vessels and heart-cavities.[2] As suggested by Robin, the oil in these cases may have exuded from the tissues as a result of post-mortem decomposition, and in one of the cases its presence in the cavities of the heart may be explained by the fact that rupture of the ventricle had occurred. Facts go to show that the etiology involves both general and local causes.

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[1] Ch. Robin, *vide* Chimie Anatomique; also Dictionnaire de Médecine (Nysten), art. Dégénération.

[2] These cases are reported in the Dublin Journal of Medical Science, first series, vol. ix, p. 411. See Stokes on Diseases of the Heart and Aorta.

Among the local causes is impairment of nutrition from obstruction of the coronary arteries. Of eighty-three cases analyzed by Quain, these arteries were calcified or obstructed in twenty-five. Atheromatous and calcareous disease of the coronary arteries especially lead to impairment of nutrition from the fact that these arteries do not communicate with each other by anastomosing branches. Embolism or thrombosis of these vessels occasions functional weakness and softening without fatty degeneration, the obstruction thus produced being sudden, instead of gradual as it is from atheroma and calcification. Pericarditis and endocarditis are reckoned as local causes. Wagner found fatty degeneration after pericarditis in 17 of 35 cases of the latter disease, and in 28 of 75 cases of valvular lesions.[1] It is often associated with enlargement of the heart dependent, not only on valvular lesions, but on emphysema and Bright's diseases. The author just referred to found it in 12 of 35 cases in which enlargement of the heart was secondary to affections of the kidneys. Of 49 cases of fatty degeneration analyzed by Dr. T. K. Chambers, the heart was enlarged in 29.

Fatty degeneration occurs in connection with certain general affections sufficiently often to render probable a causative relation. It occurs in connection with the tuberculous and the carcinomatous cachexia; it is stated to occur also in the essential fevers, and in cases of pyæmia or septicæmia. It is incident to the toxical effects of phosphorus, arsenic, and some other poisons. Alcoholism undoubtedly has a causative influence. Of the cases in which this lesion is found after death, in a large proportion intemperate habits, as regards alcohol, will be found to have existed. In the great majority of cases fatty degeneration occurs in middle or advanced age. It is rare in early life. It is much oftener found in male than in female subjects.

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[1] Raynaud, *op. cit.*

SYMPTOMS AND PATHOLOGICAL EFFECTS OF FATTY GROWTH AND DEGENERATION.

Although the different forms of fatty disease differ pathologically, their general effects are similar. They induce alike weakness of the heart, and symptomatic phenomena due to enfeebled circulation. But they by no means induce these effects in an equal degree. A considerable accumulation of fat upon the heart, as stated already, may exist without giving rise to symptoms which point to cardiac disease. The effects are more marked if the fatty growth be interstitial, or if the organ become infiltrated with fat. They are still more strongly marked when atrophy of the muscular tissue has occurred to much extent. Each variety, of course, is important, in proportion to its diffusion and amount; but the microscope not infrequently shows considerable degeneration in hearts in which disease had not been suspected either from the symptoms during life or the general appearance after death. I have met with a case in which disease of the heart was not suspected, and apparently the person was in good health, up to the occurrence of rupture, the heart being found, after death, to have undergone extensive fatty degeneration. The account which will be given of the symptoms and pathological effects, will have reference especially to fatty degeneration, as distinguished from fatty growth.

The symptoms referable directly to the heart and circulation are not distinctive. The pulse, if the heart be not very greatly weakened, may be natural in frequency and perfectly regular. It will lack force; but the differences in this respect are so great among healthy persons, owing to a variety of circumstances, that this quality of the pulse does not possess much significance. Notable slowness of the pulse has been observed, the number of pulses per minute falling to twenty or thirty, and even lower. They have been observed as low as eight and nine per minute.[1] In an advanced stage of disease, intermittency, irregularity, and great frequency, conjoined with extreme feebleness, are liable to occur; these characters, however, belong equally to the pulse in cases of dilatation, and do not indicate, specially, fatty disease. The pulse and systemic circulation will, of course, furnish manifestations of cardiac weakness more marked in proportion as the disease affects the left ventricle. General dropsy rarely occurs as a result of merely weakness of the heart, exclusive of valvular lesions and dilatation. Sense of oppression at the præcordia, palpitation, and a tendency to syncope, are symptoms referable directly to the heart and circulation. I have also observed a notable degree of capillary congestion of the extremities.

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[1] *Vide* Memoir on "Slow Pulse," by Mr. Richardson, Dublin Quarterly Journal, vol. xiv. Dr. Bellingham remarks, with reason, that this abnormal slowness involves an abnormal cerebral condition superadded to the heart-affection

Dyspnœa on exercise is present in proportion as the right ventricle is weakened. But this symptom does not represent exclusively weakness of this ventricle. If the contractions of the left ventricle be feeble and incomplete, the left auricle necessarily becomes distended, and pulmonary congestion ensues. Hence more or less dyspnœa is present when fatty degeneration is confined to the left ventricle, provided its muscular power be considerably impaired.

The foregoing symptoms are common to all affections which impair the power of the heart's action. They point only to some cardiac trouble involving diminished power of the organ. Considered alone, they are not diagnostic of the affections under consideration.

Certain symptoms pertaining to the nervous and the respiratory system have been supposed to be highly significant of fatty degeneration of the heart. The occurrence of seizures resembling apoplexy, appears to have been first observed by Cheyne.[1] Subsequently cases were reported by Adams, Law, and Stokes, of Dublin. These attacks are characterized by the sudden loss of consciousness, and recovery without paralysis. In some cases they recurred frequently, in the end proving fatal, and on dissection the brain presented no morbid appearances adequate to explain their occurrence. The heart had undergone extensive fatty change, and the cavities were dilated. Among my recorded cases are two in which pseudo-apoplectic seizures were observed in conjunction with symptoms and signs denoting fatty degeneration of the heart, recovery taking place, in each case, as regards the affection of the nervous system. In one of these cases the patient was for three days in a semi-comatose condition.

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[1] Dublin Hospital Reports, vol. ii. Also Stokes on Diseases of the Heart and Aorta, Am. ed., p. 319.

The nature of the pathological relation existing between attacks of pseudo-apoplexy and the cardiac affections under consideration, with our present knowledge, can only be conjectured. That it consists in disordered cerebral circulation due exclusively to the weakened condition of the heart, is not probable, inasmuch as the muscular power of the organ is equally reduced in cases of simple and complicated dilatation. It is more rational to suppose that some intermediate morbid conditions are involved; conditions not dependent on cardiac disease, but associated, more or less frequently, with it. Fatty disease or calcification of the cerebral arteries, lesions often associated with fatty degeneration of the heart, may, in conjunction with the latter, occasion these attacks. In one of the cases reported by Stokes, calcification of the cerebral arteries existed in a notable degree.

A peculiar aberration of the respiratory movements was described by Cheyne, and afterward by Stokes as characteristic of great weakness of the heart incident to fatty degeneration. This symptom is thus described by the distinguished author last named: "It consists in the occurrence of a series of inspirations, increasing to a maximum, and then declining in force and length, until a state of apparent apnœa is established. In this condition the patient may remain for such a length of time as to make his attendants believe that he is dead, when a low inspiration, followed by one more decided, marks the commencement of a new ascending and then descending series of inspirations. This symptom, as occurring in its highest degree, I have only seen during a few weeks previous to the death of the patient." This aberration of respiration has, however, been observed in connection with hypertrophy of the left ventricle from valvular disease without fatty degeneration.[1]

Fatty degeneration of the cornea, giving rise to the appearance well known as the *arcus senilis*, has been observed in cases of fatty degeneration of the heart. It has been supposed to be valuable as a symptom from its frequent coincidence with the latter. It may be regarded as at least denoting a condition of the system favorable for fatty degeneration, and, taken in connection with the symptoms and signs relating directly to the heart, it is not without diagnostic import. The degree of significance which belongs to it depends, of course, on the frequency or constancy of the association. The association is certainly not constant enough to constitute it a diagnostic symptom of fatty degeneration of the heart, exclusive of physical signs and other symptoms; and, on the other hand, its absence is by no means to be considered as proof that the cardiac affection does not exist.[1]

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[1] On the Diseases of the Heart and Aorta, Am. ed., p. 340.

According to Walshe, failure of the sexual inclination and power is to be included among the pathological effects. He states that in one of the best marked cases of the disease which he ever saw, clinically speaking, in a person under forty-five years of age, he was consulted for impotency, without reference to the cardiac symptoms.

Paroxysms of pain, constituting *angina pectoris*, may occur in conjunction with fatty degeneration. No special pathological relation, however, exists between these affections inasmuch as angina pectoris is associated with other organic lesions of the heart. Being common to different cardiac affections, I shall treat of it under the head of functional disorders of the heart.

The pathological effects of fatty growth and degeneration are serious in proportion as they involve structural change, or, in other words, atrophy and impaired consistence of the walls of the heart. Hence, fatty degeneration is the form of disease which is especially attended with notable disorder and danger. The muscular substance lost is replaced by the deposit of fat, which makes good the volume of the heart, but, of course, without supplying, in any measure, the loss as regards the function of the organ. It may prove fatal, by leading to rupture. Sudden death is liable to occur in an attack of syncope; the cavities becoming overloaded, and the walls too feeble to propel the blood, the movements of the organ are suddenly arrested. Exclusive of these cases, a fatal result is generally not due directly to the cardiac affection, but to the concurrent effects of associated or superinduced pathological conditions. This, however, by no means renders the existence of fatty degeneration unimportant, either as regards diagnosis or treatment. As stated by Stokes, the presence of an amount of structural change, not sufficient to give rise to well-marked symptoms of cardiac disease, may serve to explain the disproportionate feebleness of the circulation, tendency to syncope, and the intolerance of bloodletting and other debilitating measures, which are sometimes observed in different affections. The probability of the coincidence of the affection under consideration, is to be taken into account in cases in which the fact cannot be positively determined.

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[1] Mr. Edward Canton, of London, first ascertained that the *arcus senilis* is due to fatty degeneration of the cornea, and advocated the opinion that, when present, under the age of 40, it is invariably associated with fatty heart, *vide* London Lancet, 1850 and 1851. As regards the frequency of its association with fatty heart, clinical experience is thus far discrepant. *Vide* Williams's Principles of Medicine, and article by Dr. Hopkins, in Am. Journ. of Med. Sci., Jan., 1853. Also, an Essay "On the Symptomatic Value of the Arcus Senilis; with a Tabulated Statement of Seventy-two Cases." By Benjamin Lee, A.M., M.D., in the Am. Med. Month., Sept., 1856.

The term *asystole* is applied by French writers to denote the incompetency of the ventricular contractions, which is incident to fatty degeneration, as well as other lesions impairing in a notable degree the power of the heart's action. I have notes of five cases of sudden death, in which the autopsy showed fatty degeneration of the heart and no other cause, these cases having come under my observation since the publication of the first edition of this work.

PHYSICAL SIGNS AND DIAGNOSIS OF FATTY GROWTH AND DEGENERATION.

The accumulation of fat is rarely sufficient to increase the size of the heart much beyond the limit of the variations in health. If, therefore, percussion show a considerable amount of enlargement, dilatation is to be inferred. Dilatation and the accumulation of fat are not infrequently combined, and the question at once arises, is the diagnosis of the latter, under these circumstances, practicable? The extent of enlargement of the heart can generally be determined with precision. By means of percussion and palpation, the space which the organ occupies can be delineated and measured. Now, if the enlargement be sufficient in extent to correspond with the associated signs and symptoms, the evidence of a fatty heart is wanting; but if, on the contrary, the signs and symptoms denote a degree of cardiac weakness out of proportion to the enlargement, a fatty heart may be strongly suspected; and, if other circumstances are present pointing to the latter, the diagnosis may frequently be made with much positiveness. The exclusion of valvular lesions is an important point in the diagnosis. If valvular lesions be not present, the coexistence of fatty degeneration is rendered highly probable by the fact of dilatation, the latter probably occurring in consequence of the former. It is to be borne in mind that the presence of valvular lesions by no means precludes the existence of fatty disease; but in cases in which valvular lesions, enlargement, and fatty growth or degeneration are combined, the diagnosis of the latter cannot certainly be made with positiveness. Fatty growth or degeneration may, however, be reasonably suspected when, under these circumstances, the weakness of the heart is greater than would be expected from the amount of dilatation.

Limiting the attention to cases in which fatty degeneration is the sole or paramount lesion, and in which the atrophic changes are sufficient to give rise to well-marked manifestations of a cardiac affection, what are the physical signs furnished by the different methods of exploration? Percussion shows moderate or no increase of the volume of the heart. This is an important negative point. The apex-beat, if felt, will be but little, if at all, removed from its normal situation. The beat, if felt, is abnormally feeble, and it will be inappreciable if the heart be greatly weakened. Impulses elsewhere than over the apex, will, in general, not be discoverable. Inspection may disclose a very feeble movement over the apex, or none whatever. The diminished force or suppression of the apex-beat will depend, of course, on the extent to which the left ventricle is affected. The sounds of the heart are weakened. The first sound, more than the second, shows abnormal weakness. It is also short and valvular, resembling, in these respects, the second sound. The greater weakness of the first sound and its altered quality, are due especially to the effect upon the element of impulsion. This element is impaired more than the valvular element, and may be suppressed while the latter remains. The first sound may be wanting, the second sound being still heard; and, finally, both sounds may be extinct. The latter obtains in cases of a very great degree of fatty degeneration.

When, in connection with these physical signs, there are present symptoms denoting a cardiac affection, viz.: feebleness, and perhaps irregularity of the pulse; palpitation and præcordial distress; dyspnœa on exercise, tendency to syncope, &c., there can be but little room for doubt that the heart is affected with fatty degeneration, especially if the patient have passed the middle period of life, if his habits of life have been luxurious and indolent, if he have been addicted to alcoholic beverages, if he have the *arcus senilis*, or if there be a tendency to obesity. The diagnosis is not difficult under these circumstances. It is less easy when the problem is to decide whether fatty degeneration exists in addition to enlargement and valvular disease; and also when the amount of degeneration is not sufficiently great to give rise to well-marked symptoms and signs of cardiac disease. The probability of the coexistence of this lesion with other affections which the physician is called upon to treat, is important to be taken into account in the interpretation of symptomatic phenomena, and the employment of therapeutical measures.

TREATMENT OF FATTY GROWTH AND DEGENERATION.

The general objects of medical treatment in cases of fatty growth and degeneration are threefold, viz., 1. To obviate and relieve the immediate effects of weakness of the heart; 2. To increase permanently the muscular power of the organ; and 3. To arrest or limit the accumulation of fat.

Of the immediate effects of the cardiac weakness incident to these affections, the more prominent are palpitation and præcordial distress, syncope, dyspnœa, and, perhaps, apoplectiform coma. These effects occur generally in paroxysms, induced by causes which either temporarily increase the habitual weakness, or which, like exercise, mental excitement, &c., overtask the power of the heart. Some of the effects, however, may be more or less constant. The means of obviating and relieving them consist of measures to augment the force of the ventricular contractions; in other words, the use of remedies which act as cardiac stimulants. These are wine or spirits, ether, and the carbonate of ammonia. They are to be given more or less freely according to the urgency of the symptoms, that is, in proportion to the degree of cardiac weakness; and they are to be continued or repeated according to the persistence or recurrence of the paroxysms. Their habitual use is indicated if the effects be constant. The particular stimulants to be selected must vary with reference to the habits of patients and the results of experience in individual cases. As regards quantity, they are to be graduated by the symptoms and by the relief afforded. It is impossible to formularize the means of fulfilling this object of treatment. Here, as well as with reference to the other objects of treatment, with a clear idea of the ends and means, the judicious practitioner will not be at a loss as regards therapeutical details. Without a proper knowledge of the pathological character of the immediate effects, serious errors of practice may be committed. Depletion by bloodletting or otherwise, and all measures tending to enfeeble still more the circulation, can hardly fail to be pernicious. In the attacks of pseudo-apoplexy, which have been referred to, whether immediately dependent on the heart or not, stimulants are not to be withheld on the supposition that the brain is congested. Cerebral congestion, it is to be borne in mind, may proceed from enfeebled power of the heart. In employing diffusible or alcoholic stimulants, the aim is not to excite the heart, but to strengthen its action. If they produce greater frequency of the pulse, the end is not attained. Their effect should be augmented force and volume of the pulse; and with this effect the frequency may be diminished and the rhythm become more regular. Digitalis may be employed tentatively, in moderate doses, with reference to great frequency, irregularity, and feebleness of the heart's action. Revulsive measures, such as stimulating pediluvia, are useful, as in dilatation, by diverting the blood from the heart, and thus diminishing, for the time, the labor of the circulation. These remarks apply to the remedial measures. It is hardly necessary to say that all the causes which either increase temporarily the habitual weakness or overtask the power of the heart are, as far as practicable, to be avoided and removed. Undue fatigue or depressing agencies of all kinds, physical and mental, violent muscular exertions, excitement, &c., belong in the category of exciting causes.

The second object, viz., to increase permanently the muscular power of the heart, is to be effected by tonic remedies, by an appropriate system of diet and regimen, and by judiciously regulated exercise. Tonic remedies are called for with a view to improvement of the appetite and digestion, if impaired or disordered. Quinia, bitter infusions, and the mineral acids, are useful with reference to this end. The preparations of iron are especially indicated if anæmia exist. An anæmic condition is to be dreaded not less in these than in other affections of the heart; in all, the symptoms are greatly aggravated whenever it coexists. Flatulency and constipation are to be relieved by suitable remedies. With reference to the proper performance of the digestive functions, wine, spirits, or beer, in moderate quantity, may generally be taken with advantage. The dietetic management is of very great importance. The end is to contribute toward the healthy nutrition of the affected organ by rendering the blood rich in nutritive materials. For this end, the articles of diet should be highly nutritious and easily assimilated. The diet should consist of as large a proportion of animal food as the digestive powers will permit. The quantity of fluids should be restricted, in order that, while the blood is enriched in quality, the vessels and heart-cavities shall not be unduly repleted, the labor of carrying on the circulation being proportionate to the mass of liquid to be circulated. Warm clothing is of importance to secure the distribution of a proper proportion of blood to the surface and extremities. Excesses of all kinds, in eating, drinking, venery, mental occupation or excitement, late hours, &c., are to be rigidly interdicted. Regular habits of life in all respects are important. Judiciously regulated exercise in the open air constitutes a part of the management not least in importance. Physical indolence predisposes to these affections; and, on the other hand, by habitual, systematic exercise, the heart is directly strengthened. Great caution, however, is to be observed in this part of the management. While judiciously regulated exercise is of great importance, injudicious excess may do much harm. The practical rule to be observed here is the same as in cases of dilatation. The patient is to be encouraged to take such exercise as he is able to take without experiencing inconvenience from dyspnœa or palpitation; in other words, undue excitation of the heart is to be avoided. As the ability to endure greater and more prolonged exercise augments, the limit may be enlarged and extended. Violent exertions are never appropriate; walking, riding, rowing, the use of dumb-bells, &c., should be the modes of exercise resorted to. Pedestrian and equestrian excursions, involving the mental interest of travelling, and hunting, and fishing, if pursued with zest, are especially to be recommended. Proper instruction and cautions are to be enjoined, as the tendency with many persons is, when once a system of exercise is undertaken, to push it to an extreme. The reader will have observed that thus far the principles of management do not differ materially from those to be pursued in cases of dilatation of the heart. The ends of management in dilatation and in fatty degeneration are, in fact, the same. The two lesions are often combined, and in treating the latter, an incidental object is to endeavor to prevent or limit the former.

The third object, viz., to arrest or limit the fatty accumulation, is peculiar to the affections under consideration. Theoretically considered, this object more clearly relates to fatty growth than to fatty degeneration. The former, in general, involves a tendency to the accumulation of fat in different parts of the body, or the adipose diathesis. The latter does not so generally involve this tendency or diathesis. That it does so to a greater or less extent is, however, probable. In fact, fatty growth and degeneration are often associated. The constitutional disposition, or, to speak more properly, the state of the blood favorable to obesity, may be controlled in a great degree by a diet adapted to this end, conjoined with habits of exercise. Fatty and saccharine substances should be interdicted, and articles abounding in amylaceous principles are to be sparingly allowed. The diet should consist of meat, bread, non-farinaceous vegetables, and certain kinds of fruit. Vegetables which are highly farinaceous, such as potatoes and rice, should be taken sparingly. By following this plan with perseverance, I have known an excessive corpulence greatly reduced, and the general health much improved. Caution is here necessary, lest the dietetic course be pushed to an extreme. The powers of the system are by no means to be lowered. A proper variety of alimentary principles is to be provided. It is only necessary that fatty substances be interdicted, and that those principles readily transformed into fat, viz., sugar and starch, constitute, relatively, a small proportion of the articles of diet. Habits of exercise, it is well known, tend to prevent the accumulation of fat. In this point of view, they are useful, in addition to their more direct effect, in increasing permanently the muscular power of the organ. Dr. Stokes thinks that exercise during summer, or in a warm climate, when it is attended by copious perspiration, is especially useful, fatty principles being eliminated in considerable quantity from the surface.

As regards the success of treatment, so far as atrophy or degeneration of structure has taken place, the lesion must be considered as incurable. It is not probable that the substance which has disappeared is reproduced. But the heart, like the other important organs of the body, may sustain a certain amount of damage, while there still remain sufficient healthy tissue and functional power for life and health; and although that which is actually lost in structure cannot be recovered, the deficiency may be made up by increasing the development and vigorous action of the normal tissue which remains. Thus, a heart more or less unsound from fatty degeneration, may perhaps be rendered more efficient than it was even before the degeneration commenced. This statement is, of course, not applicable when the unsoundness is considerable. But assuming that a certain degree of weakness is inevitable for the remainder of life, if the progress of the structural affection can be stayed, and the capabilities of the organ developed and maintained, the condition of the patient may not be serious, even with a considerable amount of unsoundness. With this view of the subject, the importance of an early diagnosis is sufficiently obvious.

The importance of recognizing fatty disease of heart in connection with inflammatory diseases affecting other organs, is not to be lost sight of. If there be grounds for suspecting the coexistence of fatty growth, and more especially fatty degeneration, depletion and debilitating measures are to be employed with great circumspection. The question as to the presence of these affections will, in fact, oftener arise in such a connection, than in cases in which attention is called to a cardiac disease exclusively.

SOFTENING OF THE HEART.

Softening of the heart has been already considered as incidental to fatty degeneration. It will be noticed in a future chapter as a result of inflammation. Exclusive of these pathological conditions, it belongs among the anatomical changes which are liable to take place in the course of the essential fevers, especially typhoid fever and typhus. Its occurrence in typhoid fever was observed by Laennec; but it was more fully studied by Louis, in the researches which established the natural history of that disease. In a certain proportion of cases of typhoid fever ending fatally, Louis found the muscular walls of the heart more or less softened. Sometimes the softening was limited to the left ventricle, and sometimes it extended over both ventricles. When the diminished consistence was marked, the walls were notably relaxed and friable; the structure was easily torn and penetrated with the finger; the organ was flaccid, collapsing by its own weight, and not preserving its natural form, but retaining, like a wet cloth, any shape in which it was placed. When incised, the cut surfaces were dry and unpolished, and the color of the muscular tissue was purplish or livid. These alterations were not accompanied by any marked change in volume, nor by any of the products of inflammation.[1]

Softening of the heart, as occurring both in typhoid and typhus fever, has also been studied by Stokes, his observations agreeing in all important particulars with those of Louis. Stokes has observed instances in which the external muscular layer of the left ventricle appeared to be converted into a homogeneous substance, all traces of muscular fibre being lost. He has also remarked an adhesive, gummy liquid, with which the affected muscular substance was infiltrated.[2]

The softening, under these circumstances, is evidently due to a process which may be called acute, for it occurs early in the disease, and is most frequent and marked in the bodies of those who have died after a short career of the fever. Examinations made soon after death show that it is not due to cadaveric decomposition, with which, without proper care, it is liable to be confounded. It may be associated with softening of other viscera, especially of the liver and spleen. The researches of Zenker have shown that softening, due to degenerative changes, also occurs in certain of the voluntary muscles, the adductors of the thigh and the abdominal recti being especially liable to become affected.[3]

Softening of the heart occurring in the essential fevers is by some attributed to inflammation of the muscular walls.[4] A more rational view is, that it depends on molecular changes incident to a morbid state of the blood, or to abnormal innervation, or to both, without involving myocarditis. As already stated, fatty degeneration is considered as sometimes taking place in the course of the essential fevers. Zenker has described other degenerative changes, namely, the substitution for the muscular substance of granular matter, and of a translucent material giving the appearance of waxy degeneration. Whatever may be the changes upon which the softening depends, it occasions, in certain cases, a degree of weakness of the heart, which renders it an important event in the progress of the disease, contributing not infrequently, to a fatal result. Clinical observation, however, furnishes evidence that softening occurs without leading to serious consequences, and that it admits of complete restoration. Softening of the heart from fatty degeneration, or other degenerative changes, rarely, if ever, remains, as a permanent lesion, after recovery from typhus and typhoid fever.

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[1] *Vide* Anatomical, Pathological, and Therapeutic Researches upon the Disease known under the name of Gastric-enterite, &c. Translated by H. J. Bowditch, M.D., 1836, vol. i.

[2] Stokes on Diseases of the Heart and Aorta. Am. ed., p. 388.

[3] For an abstract of a monograph on this subject, by Dr. P. A. Zenker, Professor of Pathological Anatomy in the University of Erlangen, see Archives de Médecine, Août et Septembre, 1865.

[4] *Vide* Raynaud, *op. cit.*

Softening of the heart does not belong exclusively to typhus and typhoid or other essential fevers. It has been observed in purulent infection of the blood, in scorbutus, purpura, and other affections. Except as developed during the progress of some general disease, it probably rarely, if ever, occurs independently of fatty degeneration or inflammation.

The symptoms and pathological effects of softening of the heart are essentially the same as in fatty degeneration. They proceed from weakness of the organ, and are commensurate, as regards their intensity, with the loss of muscular power incident to the lesions. The pulse, in both cases, represents but imperfectly the degree of cardiac weakness. As regards the force of the pulse, exclusive of the systole of the left ventricle, much depends on the degree of resistance in the vessels to the passage of blood, in other words, on the amount of arterial tension. Feebleness of the pulse, therefore, even when the feebleness is great, is by no means distinctive of this lesion. In cases of fever, the symptoms and pathological effects of cardiac softening are so intermingled with the phenomena pertaining to the febrile disease that it is impossible to isolate them. Functional weakness of the heart, without softening, is sufficiently common in typhus and typhoid fever, and the feebleness of the circulation, particularly as denoted by the pulse, may be as great in the one case as in the other. The diagnosis of softening, in short, cannot always be made with positiveness. But, with the aid of physical signs, the occurrence of this lesion may be determined, in some cases, with considerable confidence. It suffices for all practical purposes to consider the physical signs and diagnosis of softening as occurring in typhus and typhoid fever. The same considerations are applicable when it takes place in other pathological connections.

The physical signs of softening of the heart in fever were first thoroughly studied, and their importance enforced by Stokes.[1] The signs are essentially those which belong to fatty degeneration, but, at the time of the original observations by Stokes, the latter affection was very imperfectly understood. To recapitulate these signs in the present connection, the apex-impulse becomes notably feeble or it is suppressed; the intensity of both sounds is diminished, but the first sound is relatively much more weakened than the second. The first sound is altered in quality and duration, becoming short and valvular, in these respects resembling the second sound; in other words, it loses those characters which belong to the element of impulsion, and is analogous to the sound of the fœtal heart. These abnormal changes may be more or less strongly marked. The first sound is sometimes extinguished, while the second sound continues to be heard, and in some instances both sounds are inappreciable. The latter indicates great weakness of the heart, and is very rarely observed. Taking place in the second week of the career of typhus or typhoid fever, when, during the early period of the disease, the impulse and sounds had been sufficiently intense, and had presented their normal characters, these signs denote either softening or simply functional weakness of the heart. Therapeutically, the indications are the same whether they are due to softening or to functional weakness, and, in a practical point of view, it is not of great importance to make the discrimination. As a matter of scientific interest, however, the differential diagnosis is deserving of attention. What, then, are the points which indicate softening? The researches of Louis show that softening is apt to occur rather early in the febrile career, at or soon after the end of the first week. Functional weakness, sufficient to give rise to the abnormal modifications of the sounds and impulse which have been described, is not likely to occur until a later period. If, therefore, the signs are present early, the presumption is in favor of softening. Functional weakness will be likely to be associated with marked general debility of the muscular system. It occurs in cases characterized by adynamia. Softening may take place when the voluntary muscles do not manifest extreme prostration. Want of correspondence, therefore, between the evidences of cardiac weakness and the condition of the general muscular system points to softening. Stokes attaches significance to the slow development of softening. The signs of the cardiac weakness, due to this lesion, are observed to become progressively but gradually marked, and then for some time steadily persist, while functional debility is liable to be rapidly induced, to vary from day to day, and is often less persistent. A point more distinctive than any other, which is available in a certain proportion of cases, relates to the results of a comparison of the heart-sounds in different situations within the præcordia. It has been already stated that the softening is limited to, or especially marked in, the left ventricle. Now, as stated by Stokes, under these circumstances, the first sound may be louder at the inferior border of the heart, where it is derived from the action of the right ventricle, than over the left ventricle. This shows that the muscular power of the latter is diminished more than that of the former, a fact which is highly significant of softening, because the causes inducing merely functional weakness are alike operative on the two ventricles. Finally, the reduction of the pulse in frequency below the normal average, which is observed at the time of convalescence in a certain proportion of cases of typhus and typhoid fever, is supposed by Stokes to denote that softening has taken place.

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[1] Dr. Stokes's original observations were made in 1837 and 1838, and published shortly afterwards. But the reader is referred to his work on Diseases of the Heart and Aorta for a full consideration of the subject.

TREATMENT OF SOFTENING OF THE HEART.

The occurrence of softening of the heart in the course of typhus and typhoid fever furnishes an additional indication for sustaining measures, viz.: alcoholic stimulants, and alimentation. The tendency to death by asthenia is increased by this complication, and, hence, the means of obviating this tendency are to be pushed with more vigor whenever there are reasonable grounds for supposing that it has taken place. These remarks are equally applicable to the treatment of softening when it occurs in other pathological connections, as in the eruptive fevers, pyemia, &c. Stokes has called attention to the importance of the physical signs of cardiac weakness in determining the extent to which sustaining measures, and especially alcoholic stimulants, are indicated in the treatment of fevers. The diminished intensity or suppression of the first sound of the heart, together with alterations in its duration and quality, constitutes a better criterion of the loss of muscular power which the organ has sustained, than the pulse or other symptoms. In fact, the pulse, as stated already, does not always represent fairly the force with which the left ventricle contracts. The evidence obtained by auscultation and palpation is more reliable. With reference to this end, physical exploration, practised from day to day during the febrile career, is of great practical value, and is too much neglected by medical practitioners. In endeavoring to decide between softening and functional debility of the heart, there may be often room for doubt, but, happily, so far as relates to treatment, the indications are the same in both cases, and, consequently, no harm results from error in this differential diagnosis. Practically, the important end is to estimate correctly, by means of the physical signs, the degree of cardiac weakness.

INDURATION OF THE HEART.

Induration of the muscular walls of the heart, sufficient to constitute an important lesion, is rare. Pathologically it involves different conditions. An instance was described by Corvisart in which the heart, when struck, sounded like a dice-box or hollow horn vessel, and yet the natural appearance of the muscular substance was preserved. The microscope, however, had not then been brought to bear on the study of minute anatomy. Portions of the walls present sometimes the firmness and appearance of cartilage. This must proceed from either the production of fibroid tissue or a morbid increase of the interstitial connective structure. Calcareous plates are sometimes formed upon the surface of the organ, and the calcareous matter may extend between the muscular fibres. I have met with a specimen in which a calcareous deposit near the base of the left ventricle extended through the muscular walls, and formed a projection, through the endocardium, into the ventricular cavity. In a case described by Burns, the ventricles were said to be so completely ossified as to resemble the bones of the cranium. Allowance is doubtless to be made for exaggeration in this comparison. In a practical point of view, induration of the cardiac walls claims only a passing notice. Not only is it extremely rare, but it is wanting in distinctive signs and symptoms. The diagnosis is impossible. It may be stated as a rule applicable, at least, to diseases of the heart, that the difficulty of diagnosis is inversely in proportion to its practical importance. This lesion affords an illustration of the rule. Induration proceeding from either of the conditions mentioned, is irremediable. It was conjectured by Laennec that the heart-sounds would be intensified by an indurated state of the walls, so as to be heard at a distance from the chest in some cases. Clinical observation, however, has shown, on the contrary, that the sounds are enfeebled. This would be expected in view of our present knowledge of the mechanism of the sounds.

CARDIAC ANEURISM.

The term aneurism was formerly applied to enlargement of one or more of the compartments of the heart, due either to hypertrophy or dilatation. This application of the term is manifestly inappropriate, and is now discontinued by most writers. Cardiac aneurism is properly a circumscribed or pouch-like dilatation occurring in one or more of the anatomical divisions of the organ. In the great majority of instances it is seated in the left ventricle. A few cases are on record in which it has occurred in the right ventricle and in the left auricle. It is a rare lesion, yet Thurman was able to collect for analysis, from various sources, accounts of seventy-four cases.[1] The aneurismal dilatation forms a tumor varying in size in different cases from that of a small nut to a sac as large as, or even larger than, the heart itself. It contains layers of condensed fibrin and various forms of coagula, like arterial aneurisms. It is sometimes lined or studded with calcareous matter. It occurs in the great majority of cases at the apex, but it may be situated at any point on the anterior or posterior surface of the ventricles, and on the inter-ventricular septum. The cavity of the sac communicates with the ventricular cavity by an aperture varying in different cases as respects form and size, being sometimes direct and in some cases sinuous. The walls of the sac, in some cases, include the endocardial and the pericardial membrane unbroken, the muscular substance having mostly or quite disappeared; in other cases there has occurred solution of continuity of the endocardial membrane. When the endocardial membrane is perforated, the lesion probably commenced by ulceration of this membrane, the other tissues undergoing dilatation from the pressure of the blood. This is analogous to the false aneurism of surgical writers. The tumor, under these circumstances, does not attain to a great size. In cases without perforation of the endocardium, the walls of the heart yield to the pressure of the blood, and become dilated in consequence of a morbid condition at the seat of the aneurism. This condition generally results from circumscribed inflammation, and consists of softening, or the substitution of new structure for the muscular tissue. The affection, when thus induced, is analogous to the true aneurism of surgical writers. It is supposed that a circumscribed abscess of the walls of the heart, or a hydatid cyst opening into the cavity of the ventricle, may lead to aneurism. This is a brief synopsis of the views held by pathologists concerning the formation of cardiac aneurism. For a fuller consideration of the subject, the reader is referred to works on morbid anatomy. Two or more aneurismal tumors have been observed in the same case. The lesion occurs much oftener in the male than in the female.

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[1] Mr. Thurman's paper was published in the Medico-Chirurg. Transactions, London, vol. xxi, 1838. The reader will find an abstract of it in Hope's Treatise on Diseases of the Heart, &c., Am. ed., 1842, p. 313. Also in Bellingham on Diseases of the Heart, part ii, 1857. In the latter, the reader will find copious references to the literature of the subject. See, also, "Des anévrysmes du coeur," par Pelvet, Thèse de doctorat, Paris, 1867.

Cardiac aneurism may be associated with enlargement of the heart by hypertrophy or dilatation, and with valvular lesions, but these affections do not uniformly exist, although present in a large proportion of cases. In the cases analyzed by Thurman, valvular lesions coexisted in ten, and were stated not to exist in eight, the whole number of cases of aneurism of the left ventricle being fifty-eight. In ten cases only of the whole number, *i. e.*, of fifty-eight, was the absence of hypertrophy or dilatation stated. Adhesion of the pericardial surfaces over the tumor takes place in some instances, and is wanting in others. Aneurismal dilatation of the mitral valve will be more properly noticed under the head of valvular lesions.

Aneurism of the heart may end in rupture and sudden death, the blood being poured into the pericardial sac, provided adhesion of the free surfaces of the pericardium have not taken place, and, if so, the opening may take place into the left pleural cavity. But, prior to the occurrence of this event, a fatal result may take place in consequence of the embarrassment of the circulation occasioned by the tumor, and by the concomitant lesions with which it is generally associated. Embolism of the arteries of the brain, or elsewhere, may result from the detachment of coagulated fibrin within the aneurismal sac.

The existence of this lesion is hardly determinable with positiveness during life. The symptoms, in the cases which have been reported, are those denoting some grave cardiac affection, but they are generally due, in a great measure, to coexisting valvular lesions or enlargement, or to both. Nor are the physical signs distinctive. The passage of blood currents into and from the sac, is likely to give rise to a murmur, which may accompany either sound of the heart, or both sounds. A friction-murmur may also be produced. But there are no circumstances which can lead the diagnostician to pronounce decisively that these signs are due to an aneurismal tumor. He may be led to suspect it, but he is never justified in deciding with any positiveness that it exists. The circumstances favoring such a suspicion are those which show that some anomalous form of disease is present. For example, as stated by Hope, an endocardial murmur may be found which is not referable to the arterial or auricular orifices by the rules of localization to be hereafter considered. Valvular lesions, as the source of the murmur, being thus excluded, and the murmur being evidently due to some organic affection, the hypothesis of cardiac aneurism is admissible; but intra-ventricular murmur is sometimes produced, not referable to the orifices, and, on the other hand, aneurismal dilatations do not always give rise to murmur. So a pericardial or friction-murmur may proceed from various conditions, irrespective of present pericarditis. The affection is one of the rare forms of disease which give rise to more doubt and difficulty, the better acquainted the practitioner is with the diagnostic signs and symptoms of cardiac lesions. He may be aware that he has to deal with some anomalous affection, but he is unable to determine its character. Here, as in other similar instances, the inability to arrive at the diagnosis is not, in a practical point of view, to be deplored; for, were the existence of cardiac aneurism determinable, the treatment would be that which is indicated by the symptoms without this knowledge. The lesion is irremediable, and the measures best suited to retard the dilatation and prolong life, are those which are applicable to cases of valvular lesions and enlargement, with which the affection is often associated.

In view of the infrequency of aneurism of the heart, a brief account of five examples which have fallen under my observation is subjoined.

CASE 1. The specimen, in this case, was presented to me without any history of the case. The patient was a female. At the apex of the left ventricle, a saccular dilatation existed of the size of an English walnut. There was evidence of pericarditis of old date. Partial adhesions existed, and over the aneurism the adhesions were strong. The walls of the aneurismal sac were thin, but firm. The muscular tissue over the sac had disappeared. The interior presented thin layers of fibrin. The walls of the ventricles had a healthy appearance, and they were neither hypertrophied, attenuated, nor softened. The mitral valves were sound. The organ had been mutilated in its removal, the auricles having been cut away, together with the aorta and the semilunar valves.

CASE 2. This case occurred in Bellevue Hospital, in December, 1865. The patient, a male, aged about 60, was a long time in hospital with hemiplegia. Death occurred from an apoplectic seizure. On examination of the head after death, a clot was found in the substance of the brain, and there was atheroma of the cerebral arteries. The heart weighed 20 oz. This increase of weight was due chiefly to hypertrophy of the left ventricle. The walls of this ventricle were an inch in thickness. At the apex of this ventricle was a sacculated dilatation of about the size of an English walnut. The inner surface of the sac was calcareous. The sac was, in fact, a calcareous box. The pericardium was adherent over the aneurism. The aortic segments were thickened and contracted. The mitral valves were sound.

CASE 3. This case also was in Bellevue Hospital, in November,

1866. The patient was a male, aged 50. He stated that he had been ill seven months, his chief ailments having been dyspnœa, vomiting, and general debility. The lower limbs were œdematous. The urine contained neither albumen nor casts. Enlargement of the heart was ascertained, and a mitral regurgitant murmur. He suddenly became comatous, and death took place six hours afterward. On examination after death there was found opacity of the arachnoid with large effusion into the subarachnoid space and ventricles. The right lung presented nothing abnormal. The left lung was compressed by the heart, and adhered to the pericardial sac. The pericardial surfaces were everywhere adherent. The heart was greatly enlarged; the weight was 2 lbs., the walls being thickened, and the cavities dilated. Projecting in front of the anterior curtain of the mitral valve was an aneurism of the size of the closed hand. The opening from the sac into the left ventricle admitted the index finger. The walls of the sac were ⅛ inch thick, and formed of fibrous tissue. The interior contained stratified fibrin. The pericardium was closely adherent over it, as over the whole of the heart. The mitral valves were somewhat thickened. The aorta was much dilated, and its inner surface presented atheroma and calcareous plates. The liver was cirrhosed, and weighed 3 lbs. The kidneys weighed 9½ oz.

Case 4. The patient in this case was an aged woman, at the Almshouse, Blackwell's Island. She was found dead in bed. In the left ventricle just below the semilunar valves was a perforation large enough to receive the forefinger. It was evidently old. The perforation opened into a sac of about the size of a filbert. Situated between the aorta and pulmonary artery, this sac communicated with another of about the same size by a small aperture, the second sac being situated behind the right auricle. The latter sac contained coagulated blood. Rupture of the second sac had taken place, and the pericardial sac was filled with blood. In this case the aneurism was of the false variety. The specimen was exhibited to the New York Pathological Society by my colleague, Prof. Sayre.

Case 5. In a specimen exhibited at a meeting of the New York Pathological Society by Dr. Robert Newman, the aneurism was situated at the upper part of the left ventricle, and measured 9 inches in circumference. It was partially filled with dense, laminated fibrin. The patient, a male, died suddenly, but there was no rupture of the aneurismal sac. The history of the case, aside from the sudden death, had not been ascertained.

Aneurism of the coronary artery is so rare, that it belongs among the curiosities of clinical experience. A single example has fallen under my observation. The patient came to Bellevue Hospital, and died suddenly in the examination-room before being admitted. On examination after death, the aorta was found to be considerably dilated, and the inner surface was extensively calcified. Considerable calcareous matter existed behind the valvular segments, and one of these was contracted. The heart was enlarged. The left ventricle was of the normal thickness, but the cavity was much dilated. The auricles were much dilated. The mitral valves were sound.

On looking into the left auricle an oval protuberance was seen, forming a tumor as large as an American walnut. This tumor was perfectly solid from calcareous deposit, and at the central portion the endocardial membrane was wanting, leaving the calcareous matter uncovered. The surface of the tumor within the auricle was smooth. This tumor contained a cavity into which opened the left coronary artery. The cavity contained a black coagulum. Supposing this aneurismal sac not to have been calcified, but to have ruptured, there would have been constant hemorrhage into the auricle. The blood, however, would not have been lost, but the effect would have been equivalent to a mitral regurgitant current.

RUPTURE OF THE HEART.

Spontaneous rupture of the walls of the heart is of rare occurrence. It may be doubted if it has ever occurred as a result purely of the violent muscular activity of the organ. In a physical point of view, a broken heart is a poetical license, exclusive of the cases in which the event is dependent on some prior morbid condition of the cardiac parietes. In the great majority of cases, it takes place in consequence of softening from fatty degeneration. It may follow extravasation into the muscular substance, the condition called by French writers apoplexy of the heart, which has been investigated fully by Cruveilhier; also, great attenuation of the walls in some cases of dilatation, circumscribed abscess, ulcerative perforation of the endocardium, and softening from inflammation. The seat of rupture, in the larger proportion of cases, is the left ventricle, either on the anterior or posterior surface, oftener on the former. It has been observed in the auricles as well as in either ventricle. Usually a single opening takes place, varying in size from a very minute aperture to a rent of considerable size; but in a proportion of about one-sixth of a series of cases, the rupture is multiple, that is, it occurs simultaneously in two or more different points. Ruptures have been known to occur at as many as five different points simultaneously. Raynaud cites a case in which, with rupture at two points in the right ventricle, there was a rupture in the left ventricle. It occurs almost always at an advanced period of life. The coexistence of hypertrophy or of aortic obstruction favors its occurrence. It is sometimes attributable to a paroxysm of anger, the excitement of coition, a cold bath, or to some unusual muscular exertion acting as exciting causes, but in a large proportion of the reported cases, the patients were in a state of repose when it took place.[1]

In two cases which have occurred in hospital practice under my observation, the rupture was of the right ventricle. One of these cases was at the Charity Hospital, Blackwell's Island, New York. The patient was aged. I had examined the chest, and found two aortic and two mitral murmurs. He was found dead in bed. The rupture was situated near the base in the right ventricle, and was in the form of a slit an inch in length. The walls of the ventricle were evidently affected with fatty degeneration.

In the other case the patient was admitted into the Charity Hospital at New Orleans, with delirium tremens. I did not see the patient during life. He died suddenly and unexpectedly, no affection of the heart having been suspected. On examination after death, a rent was found at the upper and anterior part of the right ventricle near the pulmonary artery. The inner layer of muscular fibres was torn over a space wider than the external opening, showing the gradual progress of the disruption from within. The heart was enlarged, weighing a fraction over fourteen ounces. The ventricular walls were not increased in thickness. The right ventricle was covered with fat, and the walls presented both the gross and microscopical characters of advanced fatty degeneration. At certain points, fatty matter appeared to have replaced the greater part of the ventricular walls, the muscular tissue being reduced to a thin layer, not more than a line in thickness. The patient was about sixty-five years of age. The previous history of the case was not ascertained.

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[1] *Vide* paper by Dr. Hallowell, of Philadelphia, giving an analysis of thirty-four cases, in the American Journal of Medical Science, 1835. For copious references to the literature of this subject, see Bellingham, *op. cit.*, part ii, 1857. *Vide* also paper by Dr. A. Dunlop, in Edinburgh Medical Journal, May, 1866, and in the New York Medical Journal, Sept., 1866.

Rupture of the heart is almost inevitably fatal, and death generally follows instantaneously. In some instances, however, life has continued for several hours, the aperture being quite small, or the escape of blood being retarded by the formation of a coagulum at the point of rupture. Cicatrization is not impossible, although exceedingly improbable. Walshe states that one case has been recorded of death from rupture in which a former rupture was discovered firmly filled by a fibrinous coagulum adherent to the wall of the heart. The mechanism by which the fatal result is produced has given rise to considerable discussion. Blood is poured into the pericardial sac with more or less rapidity according to the extent of the rupture. But this sac will not contain sufficient liquid for death to be referred to the hemorrhage alone. Paralysis of the heart from the mechanical compression of the accumulation of blood within the pericardial sac is doubtless the immediate cause of death.

I have already referred to a case in which rupture took place, the heart being affected with fatty degeneration, and the patient complaining of no symptoms referable to the heart prior to the occurrence of the rupture. This patient was about sixty years of age. He had a healthy aspect, and was accustomed to pretty active exercise. The rupture was preceded by intense pain referable to the præcordia, for which large doses of opium were given. The seat of the rupture was the anterior surface of the left ventricle. This case was in the practice of Dr. C. D. Smith. A case was reported by Dr. W. B. Lewis to the New York Pathological Society, in which rupture occurred in an attack of severe pain resembling angina pectoris. This occurrence of severe pain preceding rupture has been repeatedly noticed by others.

Time and opportunity are seldom offered for an investigation with reference to diagnosis. If life be prolonged for some hours, the symptoms are those which denote syncope with præcordial distress, and coma may ensue before dissolution. Happily, here, as in other instances in which a positive diagnosis is unattainable, it would not, if attainable, affect the treatment. The indications derived from the symptoms alone are those which would be furnished by the knowledge of the accident which has occurred. Death occurring suddenly, or a few hours after the sudden development of alarming syncope, in a person advanced in years, who had previously presented evidence of cardiac disease, and especially of fatty degeneration, warrants a strong suspicion of rupture.

Rupture of the valves of the heart, or of the tendinous cords and papillary muscles, falls more appropriately under the head of valvular lesions than in the present connection. Rupture or perforation of the interventricular septum will also be more conveniently noticed in the next chapter.

Heteromorphous Productions.—The various morbid productions which, from their being foreign to the structure of the part in which they are seated, are distinguished as heteromorphous, occur very rarely in the walls of the heart. They are, however, sometimes observed. Specimens of morbid growths or tumors, belonging to the classes lipoma or fatty, fibroma or fibrous, myoma or fleshy, and syphiloma or gummy, are occasionally met with. As stated by Raynaud, from their infrequency these are interesting chiefly as pathological curiosities. They are unattended by distinctive symptoms or signs, and they accordingly defy diagnosis. In a practical point of view, therefore, they have but little importance. Few organs in the body are more exempt from these, and other heteromorphous productions, than the heart. So slight is the probability of their existence in any individual case, that they are scarcely to be taken into account in the investigation of cardiac affections which are evidently anomalous. This statement will apply to tubercles. Miliary tubercles are sometimes observed upon the surface of the heart; but collections of the product known as yellow or cheesy tuberculous matter are amongst the rarest of pathological curiosities.

Of the occurrence of hydatid cysts, according to Raynaud, there are at least thirty cases on record. The escape of hydatid tumors into the cavities of the heart is one of the causes of sudden death; and the discharge of echinococci into these cavities is one of the sources of embolism of the arteries of either the pulmonary or systemic circulation.

Carcinoma of the heart is stated to be never primary. It may be secondary to cancer in other parts, or the heart may become affected apparently by an extension of the carcinomatous growth from the lungs or mediastinum. In the cases in which the latter explanation is not applicable, the point of departure may be either the endocardium, the pericardium, or the substance of the heart. I have met with a remarkable case in which the heart was imbedded in a mass of cancer, the muscular substance, however, remaining unaffected. A brief account of this case is as follows:

Mr. G., aged about 25, son of an eminent physician in New York, consulted me, in the summer of 1867, for want of breath on exercise. At this time his aspect was healthy; there was no emaciation nor pallor, and his muscular strength was unimpaired. The pulse was frequent, being 100 per minute. An examination of the chest led to the discovery of nothing to account for the want of breath on exercise. Some weeks afterwards, I was requested to visit him. He was now confined to his room; he was unable to lie down on account of dyspnœa, and there was considerable anasarca. The urine had been repeatedly examined for albumen with a negative result. On exploring the chest, I now found flatness on percussion, on the anterior aspect of the chest, over a space corresponding in situation, size, and form, to the pericardial sac when filled with liquid. This space extended beyond the nipple on the left side, and two or three inches beyond the right margin of the sternum. Within this space there was absence of respiratory murmur. There was no pericardial friction-murmur. The sounds of the heart were feeble and distant. There was no cardiac impulse. Laterally and posteriorly, resonance on percussion and vesicular respiration extended to the base of the chest. These signs appeared to point to chronic pericarditis with effusion. From this time to the date of his death, a period of about two and a half months, I examined the chest repeatedly, finding always the same signs. The anasarca became very great, but it was notably diminished repeatedly, sometimes by elaterium and sometimes by diuretics. Shortly before his death there was much improvement as regards the dyspnœa and dropsy. He was able to lie down, and even went out of doors. A few days before his death, however, the dyspnœa returned in an increased degree.

On examination after death, pleuritic effusion with recent lymph was found on the right side. The attack of pleurisy accounted for the increased dyspnœa during the last few days of life, and was probably the immediate cause of death. The space in which flatness and absence of respiratory murmur had been observed, was occupied by a mass of cancer, a portion of which had the character of scirrhus, and in portions softening had taken place. There were several collections of softened matter of the consistence of thick serum. The heart was imbedded in, and closely adherent to, the cancerous mass, the weight of which was estimated to be from two to three pounds. The heart was diminished in bulk, but the muscular structure presented a healthy appearance. It seemed wonderful that the action of the heart should continue when surrounded by such a quantity of morbid growth. The chest only was examined after death.

The youth of the patient is a noteworthy point in this case. It is stated, however, that cancer of the heart occurs in the young, Billard having met with a specimen in an infant three days old.[1]

Wounds of the heart, belonging to surgery, do not claim consideration in this treatise.[2] There are, however, two points of view, in which they are interesting alike to the physician and the surgeon. One of these is the tolerance of foreign bodies imbedded in the heart; the other is the ingress of foreign bodies to the heart through the systemic veins. A specimen of great interest, in both these points of view, was exhibited at a meeting of the New York Pathological Society, November 28th, 1866, by my friend and colleague, Prof. F. H. Hamilton. In this specimen a rifle-ball is completely encysted in the muscular wall of the right ventricle at the apex near the septum. The patient, when a boy, was shot accidentally, the ball entering the chest near the clavicle on the right side. He recovered in six weeks, and died twenty years afterward with pneumonia. An examination of the parts, which are in Prof. Hamilton's possession, show an aneurism of the subclavian artery, the sac being of about the size of an American walnut, this sac communicating with the internal jugular vein, and containing a cup-like depression corresponding to the size of the rifle-ball. The ball undoubtedly escaping into the jugular vein, passed to the right auricle and thence into the right ventricle, where, becoming fastened by getting beneath the trabeculæ, it made its way into the muscular wall and remained, occasioning little or no appreciable inconvenience.[1]

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[1] Raynaud, *op. cit.*

[2] The reader will find a section devoted to this subject in Art. *Cœur*, by Raynaud, in Nouveau Dictionnaire, tome huitième, Paris, 1868, page 525. See also Analysis of Cases of Wounds of Heart by Fire-arms, by Dr. Samuel S. Purple, in the New York Medical Monthly, May, 1855.

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[1] A report of this unique case is contained in the forthcoming "Surgical Memoirs of the War," published by the United States Sanitary Commission, edited by Prof. Hamilton.

CHAPTER III.

LESIONS AFFECTING THE VALVES AND ORIFICES OF THE HEART.

Aortic lesions—Mitral lesions—Primary effects of valvular lesions on the circulation—Points to be observed in post-mortem examinations—Pathological processes involved in the production of valvular lesions—Symptoms and secondary pathological effects of lesions affecting the valves and orifices of the heart—Symptoms and pathological effects referable to the heart—Enlargement of the several portions of the heart in relation to mitral, aortic, tricuspid, and pulmonic lesions, respectively—Pain, palpitation, the pulse, venous turgescence, and pulsation—Symptoms and pathological effects referable to the circulation—Cardiac dropsy—Arterial obstruction by emboli—Symptoms and pathological effects referable to the respiratory system: Dyspnœa, cardiac asthma, cough, muco-serous expectoration, hæmoptysis, pulmonary apoplexy, œdema, bronchitis, pneumonitis, pleurisy, and emphysema—Symptoms and pathological effects referable to the nervous system: Apoplexy, paralysis, arterial obstruction, defective supply of blood to brain, pseudo-apoplexy, cephalalgia, vertigo, tinnitus aurium, &c., sleep, mental condition—Symptoms and pathological effects referable to the digestive system and nutrition: Hepatic congestion, nutmeg liver, portal congestion, enlargement of liver, cirrhosis, indigestion, gastrorrhagia, enterorrhœa, melæna, hemorrhoids, enlargement of spleen, nutrition—Symptoms and pathological effects referable to the genito-urinary system: Congestion of kidneys, diminished secretion of urine, albuminuria, structural degeneration of kidney or Bright's diseases—Generative functions—Symptoms and pathological effects referable to the countenance and external appearance of the body: Lividity, expression, anæmia, capillary congestion, erythema, bloodless fingers.

LESIONS of the valves or orifices of the heart, or *valvular lesions*, are present in a very large proportion of the cases of organic disease of this organ which come under the cognizance of the physician. In addition to the intrinsic interest which belongs to them as subjects for clinical study, they are important as standing in a causative relation to other cardiac lesions, more especially enlargement of the heart, and from their pathological effects in other parts of the body. They are important as sustaining a relation of dependence to other diseases, particularly acute rheumatism, a relation which has been established by modern researches. In connection with physical signs, and as exemplifying the wonderful precision of diagnosis, which has resulted from the application of auscultation, the clinical study of these lesions is highly interesting. Inquiries, with respect to their origin and mode of production, involve pathological points of much interest and importance. To the latter, brief reference will alone be made, a full discussion of them being inconsistent with the practical objects of this work. The various morbid appearances incidental to the lesions will be summarily considered, a lengthened description belonging more appropriately to works on pathological anatomy. In treating of valvular lesions, the main object will be to show their immediate and remote effects, the symptomatic phenomena to which they give rise, their physical signs and diagnosis, and, finally, the indications for treatment. The physical signs of these lesions consist of abnormal modifications of the natural heart-sounds, and also of superadded, adventitious sounds distinguished as *murmurs*. The importance of the latter, and the various considerations connected with their diagnostic application, require that they should be treated of at some length.

Lesions of the valves and orifices of the heart, exclusive of those which are congenital, are seated as a rule in the left half of the organ; that is to say, in the great majority of cases they are either mitral or aortic. The tricuspid and pulmonic valves and orifices rarely become affected after birth. Still more infrequently do the latter present lesions so extensive as are often found in the corresponding situations in the left side of the heart. When they occur, they are generally, but not invariably, associated with either mitral or aortic lesions. It is a curious fact that the lesions of fœtal life, giving rise to the congenital malformations which will be noticed in a subsequent chapter, affect by preference the right side of the heart, reversing the rule which obtains after birth. The changes which the valves and orifices present in different cases, vary greatly in degree and kind, giving rise to a great diversity of morbid appearances. As before remarked, a full description of these belongs more appropriately to the works on pathological anatomy, and to these the reader is referred. I shall content myself here with a brief account of aortic and mitral lesions. Pulmonic and tricuspid lesions will be considered in connection with the pathological effects of valvular lesions, and also in treating of congenital malformations.

AORTIC LESIONS.

Lesions affecting the aortic valves may be limited to one or two of the semilunar segments; but in most cases all are more or less affected, although rarely to an equal extent. Thickening of the segments is one of the most frequent of the morbid changes. Of the recorded appearances in 55 cases, which I have before me, thickening, due either to exudation or morbid growth, or both, is noted in 27, and doubtless it existed, more or less, in other of the cases. A certain amount of thickening may exist, the size of the segments being unaffected. The function of the valves will not then be impaired, save that their expansion will be somewhat sluggish, and the intensity of the aortic second sound of the heart must be in some measure diminished. The thickness, too, of the segments will diminish somewhat the calibre of the aortic orifice, but not sufficiently to give rise to any appreciable morbid effects.

The segments are often contracted as well as thickened. In proportion to the amount of contraction, their function, as valves, is impaired; in other words, they are rendered more or less insufficient or incompetent. Consequently a regurgitant stream of blood takes place at the time when the second sound of the heart is produced; that is, a current of blood is forced backward from the aorta into the ventricle, by the recoil of the arterial coats. This aortic regurgitant stream or current, as regards size or quantity, will, of course, be in proportion to the amount of insufficiency of the valves. At the same time, the aortic second sound of the heart must be weakened by the diminished size of the valves as well as by the thickening. If the thickening and contraction of the segments exist alone, that is, without other valvular lesions, the result is aortic regurgitation, with, perhaps, an inconsiderable amount of obstruction to the onward or the aortic direct current of blood. The segments in some cases are so much shrunken, that there seem to be only the rudiments of the valves. One, or two, or all three of the segments may be more or less thickened and contracted.

The thickened segments are, in general, more or less rigid. One, or two, or all of the segments may remain permanently expanded in consequence of rigidity. Projecting thus into the orifice, the onward current of blood is interrupted and the orifice is diminished. Great contraction of the aortic orifice is caused by a permanent expansion of the segments and adherence together of their free margins. I have met with two specimens in which the aortic orifice was, in this way, diminished to the size of a goose-quill, and one specimen in which the orifice was of the size of a crow's-quill. Stokes has described a specimen in which the orifice was so small as to admit only of the passage of a fine probe. A similar specimen is in the cabinet of the Boston Society for Medical Improvement. It is remarkable that, in the two latter cases, the lesion occasioned so few symptoms that disease of the heart was not suspected prior to the occurrence of an acute pulmonary disease which, in each case, terminated life. This fact shows the wonderful latency, in some cases, of aortic lesions which occasion an extreme degree of obstruction. The thickness is sometimes limited or chiefly confined to the situation where the segments are in juxtaposition. The effect is to cause the two segments near this point to project and remain expanded. And, as a further effect, the division between the two segments is sometimes broken, and they are fused together. It is not easy, always, in these cases, to determine whether this fusion is the result of disease or a congenital anomaly. Three examples of this kind are among the recorded cases before me. Of course, permanent expansion of the segments destroys the aortic second sound of the heart.

Rigidity of the valves from calcareous deposit is of frequent occurrence. The segments are sometimes expanded, and being incrusted with calcareous salts, appear to be completely petrified. This was formerly called ossification. Thus, Hope describes a specimen in which the "valves, in their closed condition, their bases and the whole circumference of the aorta, were nearly converted into a solid, immovable mass of bone." A deposit of calcareous salts at the base of the segments, on their arterial aspect, is quite common. Calcareous masses here cause the segments to project, and in proportion to the amount of deposit, diminish the size of the aortic orifice, weakening also the second sound of the heart. Less frequently the calcareous deposit is on the ventricular aspect of the valves. In connection with rigidity of the valves, the sinuses of Valsalva are apt to be dilated, sometimes forming pouches of considerable depth.

Another variety of lesion consists in the presence of warty excrescences or vegetations. These are generally attached at or near the free border of the segments, on their ventricular aspect. They may be few or numerous, and they vary in size from that of a pin's head to an American walnut. I have met with a specimen, in which, depending from one of the segments, was a mass of the size last named. In another specimen, to the much contracted segments were attached vegetations an inch in length, which must have passed to and fro with the direct and regurgitive currents of blood.

These vegetations appear, in some cases, to be tumors or outgrowths, and in other cases to consist of exuded or deposited fibrin; or, again, they are morbid growths, to which coagulated fibrin is attached. They are sometimes firmly adherent, and sometimes so loosely attached that it is evident they are liable to be separated by the force of the current of blood during life, and become emboli or plugs which obstruct the circulation in the arteries of the brain, or of other parts of the body to which they are transported.

The valves sometimes become abnormally attenuated from atrophy. Rupture, under these circumstances, is liable to occur. This is most apt to take place near the free border, the latter becoming separated, to a greater or less extent, from the remainder of the segment, leaving a slit-like aperture. Sometimes there are numerous small perforations, the segments presenting a cribriform appearance. Perforations may occur at any point when the valves are softened and perhaps at the same time thickened. In one of the cases in my collection, a segment presented a hole as large as a crow's-quill. In another case there was a rupture near the free border extending a third of the width of the valve, and the valve, hanging downward, formed a kind of pouch, which contained a soft coagulum of the size of a pea, the other segments being normal.

Dr. William Gull has reported a case in which one of the valves became retroverted, in connection with rigidity of the artery behind it, that is, in the corresponding sinus of Valsalva. From the history of the case it was inferred that the sudden occurrence of the retroversion was indicated by symptoms analogous to those of rupture of the valves from violence.[1]

Obstruction to the free passage of blood through the aortic orifice, may be caused by contraction of the aorta, the valves being sound. On the other hand, the orifice is not infrequently dilated in connection with dilatation of the aorta, and the valves not being enlarged in proportion, they are insufficient, although normal, aortic regurgitation taking place.

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[1] *Vide* Guy's Hospital Reports, third series, vol. v, p. 305; also American Journal of Medical Sciences, Jan., 1860, p. 195.

Finally, rupture of the aortic valves may take place from the violent action of the heart. Of course atrophy of the valves, or softening, or the brittleness caused by calcification, would favor this accident; but it occurs, although very rarely, the valves being healthy. A striking instance occurred in 1859 in the case of a New York policeman, the accident being caused by a violent conflict in making an arrest. Grave symptoms referable to the heart supervened, and death took place four weeks after the conflict. The specimen, in this case, which was presented to the New York Pathological Society by Prof. Henry B. Sands, showed two of the aortic segments extensively torn away at their base. The endocardium was detached for a little distance below one of the segments, and a perforation existed through which a small probe could be passed from the left ventricle into the left auricle. The other segment presented a normal appearance. Fibrinous vegetations, easily removed with the handle of a scalpel, were abundant upon all the roughened surfaces presented by the torn valves. Aside from the ruptured valves, the heart was healthy.

Lesions in the aorta, extending, to a greater or less extent, above the valves, are, in the majority of cases, associated with lesions of the aortic orifice. The artery may be dilated or contracted. Its inner surface may present a granular or velvety appearance. The most frequent of the morbid appearances here observed are those which are embraced under the names atheroma and calcareous deposits. The frequency with which these lesions are associated with those affecting the aortic valves and orifice render it probable that the same causative influences are concerned in the production of the lesions in both situations. It is, however, to be considered that atheromatous or calcareous changes are found in most bodies after forty years of age, and that these changes are often present without being accompanied by valvular lesions.

MITRAL LESIONS.

Lesions at the mitral orifice are essentially the same as aortic lesions, the points of difference relating chiefly to the differences, as regards form and arrangement, of the mitral valves. The mitral curtains, like the aortic segments, are often thickened and contracted. They may be thickened, and still be competent to perform their function, namely, preventing the reflux of blood, with the ventricular systole, into the left auricle. If, however, they be contracted to much extent, they fail to perform this function, and they are then said to be insufficient. As a result of their insufficiency, a retrograde stream or current, from the ventricle into the auricle, takes place with each systole of the ventricle, the quantity of blood thus regurgitating, of course, being proportionate to the extent of the mitral insufficiency.

Other kinds of lesion occasion insufficiency of the mitral valves. Rupture of the tendinous cords connecting the free margins of the valves with the papillary muscles is one of these. The rupture may be due to softening from atheroma, or brittleness from calcification, and it may be caused by violent action of the heart without any prior disease of the cords. Of 39 recorded cases of mitral lesions, which I have before me, in 4 insufficiency was due to this lesion. In one of these cases the anterior mitral curtain was completely detached from its connection with the papillary muscles, and must have flapped to and fro through the auricular orifice with the mitral direct and the mitral regurgitant current of blood. In this specimen, at the free extremity of the curtain was a projecting, sharp-pointed stalactite which, in the to and fro movements of the valve, had perforated the posterior curtain. The ruptured tendinous cords sometimes disappear; but in other cases the broken ends remain, and present a bulbous appearance from the deposit of coagulated fibrin. In one of my specimens the mass of fibrin at the end of a ruptured cord was of the size of a medium sized pea, and in another it was of the size of a bean. These masses, by another rupture of the cords to which they are attached, may constitute emboli. Shortening of the tendinous cords or of the papillary muscles are other lesions giving rise to mitral insufficiency. To these are to be added adhesions of the curtains to the walls of the ventricle, and atrophy with perforation of the curtains. Insufficiency may also be due to enlargement of the mitral orifice in connection with dilatation of the left ventricle, the valves being sound, but, under these circumstances, not protecting against regurgitation.

On the other hand, certain lesions, either with or without insufficiency of the valves, produce more or less contraction of the orifice, and consequent obstruction to the direct current of blood from the auricle to the ventricle. The deposit of calcareous salts at the base of the valves has this effect. The calcareous deposit is sometimes so abundant in this situation as to reduce the auriculo-ventricular orifice to a small aperture. The orifice in some cases is encircled by a bone-like ring, and in these cases, if the valvular curtains be but little, or not at all affected, while there is obstruction in proportion to the amount of contraction, regurgitation does not take place. But frequently the valves are also affected, so that there is regurgitation as well as obstruction. The calcareous deposit occurs oftener on the auricular than on the ventricular side of the orifice. Vegetations and warty excrescences, such as are found at the aortic orifice, occur also in this situation. They may be attached either at the base of the mitral valves, or at their free borders. At the base they occasion more or less contraction. At the borders, by preventing coaptation of the valves, in the ventricular systole, they occasion insufficiency. Here, as at the aortic orifice, they are sometimes firmly and sometimes loosely attached; and, if loosely attached, they may be washed away with the current of blood and become emboli.

But the most frequent mode in which contraction of this orifice is produced is by the union of the curtains at their sides, leaving a narrow slit through which the blood passes from the auricle into the ventricle. This has been distinguished as the button-hole contraction. Viewed from the interior of the auricle, the mitral curtains thus united form a funnel-shaped cul-de-sac protruding into the ventricular cavity. The orifice in this variety of lesion is often so small as not to admit the end of a finger, and I have met with a specimen in which it was not larger than a crow's-quill. The curtains in this variety may be either flexible or rigid. They are sometimes incrusted with calcareous salts, and rendered thereby solid like bone. Of the 39 recorded cases of mitral lesions before me, in 16 this variety existed. The button-hole contraction may, or may not, involve insufficiency of the valves. This variety of lesion is particularly interesting with reference to the mechanism of one of the cardiac murmurs to be considered in the next chapter.

Aneurism of the mitral, as well as of the semilunar valves, is another variety of lesion. Circumscribed dilatations forming pouches, which vary from the size of a pea to that of a walnut, constitute the lesion so called. Several aneurismal dilatations in the same valve have been observed, but generally the aneurism is single. The tumor always projects toward or into the auricle. The opening may be larger than the bottom of the sac, or it may be small. The sac may contain layers of fibrin, and soft coagula. The walls sometimes consist of the membranous structures of the valve unbroken, and sometimes perforation of the inner lamina has taken place; the distinction between false and true aneurisms is thus maintained here as in aneurismal dilatations of the walls of the heart. Sooner or later, if life continue, rupture of the sac ensues, and a regurgitant stream then takes place from the ventricle to the auricle at the point of rupture. Aneurism of the valves never occurs in the right side of the heart, that is, at the pulmonic and the tricuspid orifice.

It is evident that in so far as the different lesions which have been noticed interfere with the play of the mitral valves, the first sound of the heart must be weakened and modified by the diminution or extinction of the valvular element of this sound. Thickening and contraction of the valves, rigidity, adhesion to each other or to the walls of the heart, rupture of the tendinous cords, and the incumbrance of calcareous masses or vegetations, lessen more or less the intensity of, or destroy that portion of the first sound of the heart which is caused by the sudden and forcible tension of the valvular curtains.

Reviewing the foregoing account of aortic and mitral lesions, it will be seen that their immediate pathological importance depends on their effects as regards blood-currents within the cavities of the heart. These effects are to be classified as follows:

First. The lesions may occasion obstruction to the onward or direct currents of blood, namely, the aortic direct, and the mitral direct current. Hence, they are distinguished as obstructive lesions. It is important to bear in mind that, according to the amount of contraction at the aortic or mitral orifice, the obstruction will vary in degree from that which is trivial to that which gives rise to serious effects.

Second. By damaging, or interfering with the function of the valves, the lesions occasion insufficiency, and thus give rise to abnormal currents of blood. These are distinguished as regurgitant currents. An aortic regurgitant current is an effect of insufficiency of the aortic valves, and a mitral regurgitant current is an effect of insufficiency of the mitral valves. Lesions giving rise to insufficiency of the valves, are distinguished as regurgitant lesions. Here, too, it is important to bear in mind that the amount of insufficiency and consequent regurgitation varies, the pathological importance being proportionate to the amount.

Third. Lesions may exist which neither give rise to obstruction nor to regurgitation, or the amount of either obstruction or regurgitation, or both, is too small to be of any immediate importance. The lesions which thus produce no effects of present consequence, as regards the blood-currents, may be distinguished as innocuous lesions. These lesions, nevertheless, give rise to physical signs which are to be considered in the next chapter. Innocuous lesions are thickening of the valves without sufficient diminution of the size to render them insufficient; calcareous deposits, not in large quantity, and so situated as not to occasion any material obstruction or impair the function of the valves; small perforations of the valves, and vegetations or excrescences of small size. Whatever pathological importance may belong to these lesions is not immediate but remote; that is, it relates to a prospective period when, by eventuating in greater changes, they have induced either obstruction or insufficiency. In the clinical study of valvular lesions, it is highly important, as will hereafter appear, to bear in mind the fact that lesions may exist, and give rise to physical signs, when, as regards their immediate effects, they are innocuous. To this important fact reference will again be made.

The primary effects of valvular lesions which are of immediate pathological importance, are then obstruction and insufficiency. Other things being equal, the degree of importance belonging to these effects is proportionate to the amount of obstruction and insufficiency. Obstruction may exist without insufficiency, and *vice versâ*, but it often happens that the lesions are such as to occasion both effects. These remarks apply indifferently to aortic and to mitral lesions. Now, aortic lesions may exist without mitral lesions, and *vice versâ*, but frequently both mitral and aortic lesions are present in the same case. Cases are greatly diversified by the different combinations of aortic and mitral lesions and their primary effects. Thus, there may be lesions of either the aortic or mitral orifice separately, which involve insufficiency without obstruction; the lesions in either situation may occasion obstruction without insufficiency; obstructive aortic lesions may be associated with regurgitant mitral lesions, or *vice versâ;* there may be obstruction, or, on the other hand, regurgitation, both at the aortic and mitral orifice, and, finally, aortic and mitral lesions may coexist, each involving both obstruction and insufficiency. These various combinations would seem to render the clinical study of the valvular lesions extremely complicated, but the application to this study of physical exploration has rendered it practicable, in most cases, to determine whether aortic or mitral lesions exist separately or combined, whether obstruction or regurgitation, or both, are produced by existing lesions, and to estimate the amount of damage which the heart has sustained. The reader will be better able to judge of the correctness of this statement after the physical signs and diagnosis have been considered.

As regards relative frequency in the occurrence of mitral and aortic lesions, in my own experience, the former preponderate. Of 271 recorded cases, in 111 the lesions were mitral, and in 72 aortic. In 66 of these cases mitral and aortic lesions coexisted, and in 14 cases only were the tricuspid or pulmonic valves the seat of lesions. The localization of the lesions in these cases was made either by post-mortem examinations, or by physical signs. Of 367 cases analyzed by Dr. T. K. Chambers, the mitral and aortic valves were affected with thickening, contraction, or morbid deposit in 121; the aortic valves were affected alône in 107; the mitral in 96; the mitral and tricuspid valves in 10; the mitral, aortic, and tricuspid in 10; the four sets of valves in 9; the tricuspid alone in 1; the tricuspid and aortic in 2; the aortic, mitral, and pulmonary in 2; the tricuspid and aortic in 2; and the aortic and pulmonary valves in 4.[1]

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[1] Decennium Pathologicum. Brit. and For. Med-Chir. Rev., vol. xii, 1853. In these cases were probably included cases of congenital malformation. The valves were affected in 367 of 2161 bodies examined.

In determining the pathological importance of lesions at post-mortem examinations, the points for observation are embraced in the following questions: Is there contraction of one or more of the orifices and if so, to what extent? Are the arterial or auricular valves sufficient to protect the orifices, and if not, how great is the insufficiency? These points are to be settled by laying open the cavities and carefully examining the orifices and valves. With a proper knowledge of the normal appearances, the existence or otherwise of contraction or insufficiency may be readily ascertained by the eye, and the amount of obstruction or regurgitation estimated accurately enough for practical purposes. The sufficiency or insufficiency of the aortic valves may be ascertained, before laying open the cavities and vessels, by resorting to the water test. This test consists in suspending the heart by hooks introduced into the aorta above the valves, having first tied the coronary arteries and opened the left ventricle by slicing off the apex of the organ, and then pouring a stream of water into the aorta. If the valves be sufficient, no water passes into the ventricle; but if there be insufficiency, the water escapes more or less freely from the opening at the apex. This test is far less reliable in its application to the mitral valves. It is applied to the latter by suspending the heart with the apex upwards, the left ventricle having been opened, and the aorta and coronary arteries tied. If water poured into the opening at the apex do not pass into the left auricle, the mitral valves are sufficient. This test demonstrates sufficiency in a certain number of cases. But if water do pass into the auricle, it does not follow that the mitral valves were insufficient during life; the conditions being so widely different in this experiment. If the orifices be contracted so as to oppose an obstacle to the blood-current, it is obvious to the eye, provided the observer have been accustomed to examine hearts in which the size of the orifices is normal. In recording post-mortem observations, it has been customary to note how many fingers may be passed readily through the orifice. This is a rough method of measurement, but in most instances it is sufficiently precise. Greater accuracy, of course, is obtained by actual measurement, either of the diameter or circumference. As standards of comparison, the average size in healthy hearts is to be determined. The numerous measurements by Bizot give the following mean results: The average circumference of the mitral orifice in the adult male is about four inches. The long diameter, according to Bellingham is about one inch. In the female, the size is somewhat less. The average circumference of the aortic orifice in the adult male is three inches; the diameter, according to Bellingham, is about an inch. In the female, the size is somewhat less. The tricuspid orifice is somewhat larger than the mitral in health; and this is true of the pulmonic orifice prior to the age of fifty. The observations of Bizot show that the orifices, as well as the heart itself, increase in size gradually from birth to old age.

Before proceeding to consider the symptoms and pathological effects of valvular lesions, their causation and the pathological processes involved in their production, claim a few words. The morbid changes embraced in the different lesions of which an account has been given, are thickening from interstitial exudation, and the formation of new tissue; morbid growths constituting vegetations; the deposit of coagulated fibrin derived from the blood within the cavities of the heart; and changes due to attenuation and calcareous degeneration.

To what extent is inflammation involved in these changes? Post-mortem examinations, when death has occurred during or shortly after endocarditis, show that the inflammation is generally limited to the left side of the heart; that it is seated especially at the valves and orifices, and that it induces changes involving exudation, formation of new tissue, morbid growths, and the deposit of fibrin. Moreover, clinical observation shows, *first*, that in the great majority of the cases of valvular lesions, the patients have at some former period of their lives had acute articular rheumatism; and, *second*, the occurrence of endocarditis in a considerable proportion of cases of acute articular rheumatism. From these facts it may be logically inferred that valvular lesions originate most frequently in endocardial inflammation.

The immediate local effects of endocarditis, as will be seen when we come to treat of that affection, are generally unimportant. During the progress of the endocarditis, and, perhaps, for many years afterwards, there may be no obvious symptoms denoting cardiac lesions. But the lesions which have their point of departure from rheumatic endocarditis, at length give rise to obstruction, or regurgitation, or both, and finally symptoms are developed which point to the heart as the seat of disease. When, thus, inflammation constitutes the first step in the production of valvular lesions, it is a bygone and remote event at the time these lesions have become of immediate pathological importance; it has long before ceased to be an active element of the cardiac affection, its products, with their metamorphoses, and the changes induced by them, having by degrees led to the existing morbid condition of the organ.

It is not certain, however, that valvular lesions, exclusive of those which are traumatic, that is, rupture of the valves or tendinous cords by the violent action of the heart, always proceed from inflammation. It is a mooted question whether atheroma and the calcareous deposit are to be considered as inflammatory products. They are secondary to those changes which do undoubtedly result from endocarditis; but they occur independently of these changes.

Deposits of fibrin, derived from the blood within the cavities of the heart, are incident to atheromatous and calcareous changes, as well as to those which are obviously inflammatory. They may proceed from mere roughness of the endocardial membrane, however caused. Simon's often-cited experiment illustrates this fact. The experiment consists in passing a thread through an artery; fibrin coagulates and adheres to the thread, presenting an appearance similar to the vegetations often observed on the valves of the heart. An excess of fibrin in the blood (hyperinosis), which occurs in all acute inflammations, and especially in acute articular rheumatism, probably favors the formation of these fibrinous deposits.

As regards rheumatism, either mitral or mitral and aortic lesions, oftener than aortic lesions, occur in those who at some former period have had that disease. Of 150 cases among those which I have recorded, in the histories of which the existence or non-existence of rheumatism, at a former period of life, is noted, this affection had occurred in 109; of 70 cases of mitral lesions, rheumatism had occurred in 55; of 30 cases of aortic lesions, rheumatism had occurred in 16; of 52 cases of aortic and mitral lesions, rheumatism had occurred in 38. In a considerable proportion of these cases, rheumatism had occurred repeatedly. The time which had elapsed after the last attack of rheumatism, varied from a few months to fifty-four years. In the larger proportion of cases, the rheumatic attack, or attacks, had been unusually serious and of unusually long duration.

Gout, and the abuse of alcoholic stimulants, are supposed to stand in a causative relation especially to aortic lesions. The development of these lesions is apt to be at a later period of life than in cases of mitral lesions. This is consistent with the fact that mitral lesions especially are consecutive to rheumatic endocarditis, whereas aortic lesions are oftener dependent on atheroma and calcareous deposit, the latter occurring at a later period of life than endocarditis associated with rheumatism.

SYMPTOMS AND SECONDARY PATHOLOGICAL EFFECTS OF LESIONS AFFECTING THE VALVES AND ORIFICES OF THE HEART.

Of the primary effects of valvular lesions, those which are of immediate pathological importance have been already considered. They are, obstruction to the passage of blood by contraction of the orifices, regurgitation or the flow of blood in a retrograde direction owing to insufficiency of the valves, these effects being produced either separately or conjointly. Hence, the lesions affecting the valves or orifices are distinguished as either *obstructive* or *regurgitant* lesions; and as all the valves or orifices of the heart may be affected either separately or in various combinations, valvular lesions may be divided after their seat and primary effects into obstructive and regurgitant lesions situated respectively at the mitral and the aortic orifice, and much more rarely, at the pulmonic and the tricuspid orifice. The secondary or remote pathological effects of these lesions, for the most part are traceable to the primary effects. The disturbance of the circulation, due to cardiac obstruction and regurgitation, singly or combined, gives rise to a great number and variety of morbid conditions and manifestations intrinsically more or less serious, and important, also, as symptoms of the heart affection. It will be most convenient to arrange these ulterior effects according to the different anatomical systems in which they occur. Pathological effects of great importance are produced in the heart itself; other effects are appropriately considered as pertaining to the vascular system, not being limited in their consequences to any particular situation; others relate respectively to the respiratory, nervous, digestive, genito-urinary systems, &c. In considering the effects after this arrangement, their relations to obstructive and regurgitant lesions seated at the different orifices will be incidentally considered.

SYMPTOMS AND PATHOLOGICAL EFFECTS REFERABLE TO THE HEART.

Valvular lesions involving obstruction or regurgitation, sooner or later, in the great majority of cases, lead to enlargement of the heart. They lead to this result by inducing over-distension of the cavities and over-excitement of the organ, as has been considered in the chapter devoted to the subject of enlargement. The enlargement may be due either to predominant hypertrophy or dilatation. The latter predominates in most instances in which the cardiac disease has existed for a long period, and proved directly fatal, *i. e.*, when death is not attributable to an intercurrent affection. The hypertrophy or dilatation is generally marked in, and may be limited to, certain portions of the heart. The enlargement commences at one of the ventricles or auricles, according to the situation of the valvular lesions, and thence extends successively over the other portions of the heart, observing a general rule of extension, exceptions to the rule, however, occurring not infrequently. The rule is expressed by the terms retro-hypertrophy and retro-dilatation; that is, the enlargement extends, as I shall proceed to show, over the different portions of the heart in a backward direction, as regards the normal or direct currents of blood.

Obstructive or regurgitant lesions at the mitral orifice induce, as a rule, first, dilatation of the left auricle; next, hypertrophy and dilatation of the right ventricle; next, dilatation of the right auricle, and finally, in most cases, more or less enlargement by hypertrophy or dilatation of the left ventricle. This is the regular order of effects upon the heart, the mechanism of which has been already described. Variations from this rule are not infrequently observed. Thus the right auricle is sometimes much more dilated than the left, when the valvular lesions are exclusively mitral; and occasionally under these circumstances, the left ventricle is found to be more enlarged than the right. In these exceptional cases, either the walls of the portions which are enlarged out of the natural order are particularly prone to enlargement, or there exist causes superadded to the valvular lesions. Thus, emphysema, coexisting with mitral lesions, will cause the enlargement of the right ventricle and auricle to preponderate much more than if the mitral lesion existed alone. In some cases, superadded causes may exist remote from the heart, which are not readily ascertained, causing enlargement of the left ventricle to preponderate, when, as a result of mitral lesions alone, this should be the cavity last and least affected. As an exceptional occurrence, the left ventricle is sometimes diminished in size, when, in consequence of mitral lesions, the other portions are enlarged. This fact is explained by the diminished supply of blood received by that ventricle when there exists much obstruction of the mitral orifice. The explanation applies only to cases of mitral obstruction, not to cases of mitral insufficiency without obstruction. In the latter the left ventricle is abundantly supplied with blood; hence, the conditions for the diminution in size of this portion of the heart are not present. Not infrequently, in cases of considerable mitral obstruction, the left ventricle is neither hypertrophied nor dilated when the left auricle, the right ventricle, and the right auricle are notably enlarged. My cases furnish several illustrations of this fact. The enlargement of one ventricle may be by hypertrophy, and that of the other by dilatation. Thus the right ventricle may be dilated, and the left hypertrophied, or *vice versâ*. The amount of enlargement of the heart as a whole varies greatly in different cases, and is not always proportionate to the amount of obstruction or regurgitation, a fact which shows the influence of causes subsidiary to the valvular lesions. Very great enlargement is sometimes associated with lesions involving only moderate obstruction or regurgitation, and, on the other hand, the heart may be but little or not at all enlarged, when there exists a marked degree of contraction or insufficiency. The mitral orifice has been reduced to the size of a crow's-quill, without notable enlargement of any of the cavities.[1] This fact also shows the importance of causes superadded to valvular lesions. As a rule, contraction of the mitral orifice, in other words, obstruction, tends to give rise to enlargement more than insufficiency or regurgitation; but the tendency is of course greater when, as is frequently the case, contraction and insufficiency are conjoined. The latter occurs in certain of the cases in which the mitral curtains become adherent at their sides, leaving a funnel-shaped canal opening into the ventricle by a narrow fissure resembling a button-hole or the chink of the glottis.

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[1] The cabinet of the Boston Society for Medical Improvement contains two specimens, illustrative of this statement, *vide* printed catalogue, pages 73 and 86.

Enlargement proceeding from aortic lesions invariably commences at the left ventricle. If the valvular lesions be exclusively aortic, this ventricle is always enlarged disproportionately to the other portions of the heart, and the enlargement may be limited to the left ventricle. An examination of the heart before the cavities are opened often suffices to show that the valvular lesions are probably aortic. Either hypertrophy or dilatation may predominate in the enlargement proceeding from these lesions. As a rule, if the lesions be of a nature to allow of regurgitation without producing obstruction, dilatation predominates; but if the lesions produce obstruction without regurgitation, hypertrophy is marked. This rule is not without exceptions, but it holds good in the great majority of cases. Thus, of 21 cases of either regurgitation or obstruction, the notes of which are before me, 3 only were exceptional. Of these 21 cases, in 13 there existed regurgitation without contraction, and in 2 cases hypertrophy was predominant, dilatation predominating in the others; in 8 cases there was obstruction without regurgitation, and in all save one hypertrophy was predominant. Aortic lesions, however, frequently give rise both to obstruction and regurgitation, and in proportion as the one or the other preponderates, dilatation or hypertrophy will be likely to be marked.[1] Elongation with diminution in size of the papillary muscles is found in a certain proportion of the cases of dilatation of the left ventricle from aortic insufficiency.

Enlargement of other portions of the heart than the left ventricle is not likely to result from aortic lesions alone, until after dilatation of this ventricle has taken place. After this cavity has become dilated, and the ventricular systole in consequence becomes implicated, the accumulation constitutes an obstruction to the passage of blood from this cavity into the left auricle; then it is that retro-enlargement begins. The right ventricle, however, does not become proportionately enlarged, unless there are concurrent causes which exert their effect especially on this ventricle. Pulmonary emphysema, coexisting with aortic lesions, may render the enlargement of the right ventricle as great, or even greater, than that of the left. Of the two auricles, the tendency of aortic lesions is to dilate, first and especially, the left, but in some instances dilatation of the right is more marked. Enlargement associated with aortic, as well as with mitral, lesions is by no means in all cases proportionate, as regards amount, to the degree of obstruction or regurgitation. Enormous enlargement is observed in cases in which the contraction or insufficiency is small; and, on the other hand, in some instances in which the obstruction must have been extremely great, the size of the heart has been found slightly or not at all increased. The latter fact is illustrated by a specimen contained in the cabinet of the Boston Society for Medical Improvement, the aortic orifice being so much contracted as hardly to admit of the passage of a small probe.[1] These facts here, as in the case of mitral lesions, show the importance of concurrent causes or morbid conditions in determining the amount of enlargement of the heart.

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[1] Statistics with reference to this rule are of little value unless the duration of the lesions be taken into account. In general, hypertrophy precedes dilatation whether the lesions be obstructive or regurgitant.—*Note to Second Edition.*

When, as is frequently the case, mitral and aortic lesions are associated, involving, in each situation, either obstruction or regurgitation, or both, the effects of the two classes of lesions are conjoined. Other things being equal, the enlargement of the heart, as a whole, is proportionately greater under these circumstances. The aortic lesions give rise to enlargement of the left ventricle, and combine with the mitral lesions in leading to enlargement of the other portions of the heart. Among cases of this description we are likely to find examples of excessive augmentation of bulk, constituting the *cor bovinum* of the old writers.

The pulmonic and tricuspid valves, as already stated, are rarely the seat of those structural changes which so often affect the valves of the left side of the heart. Valvular lesions seated in the right side, when they occur, are usually, but not invariably, associated with mitral or aortic lesions, either separately or combined. Their effects upon the heart are similar in kind to those of lesions seated in the left side, the point of departure for enlargement being the right auricle in cases of tricuspid obstruction or regurgitation, and the right ventricle in cases of pulmonic contraction or insufficiency. Examples of great enlargement of the right ventricle are observed in connection with congenital contraction of the pulmonary artery. Tricuspid regurgitation occurs not infrequently without, strictly speaking,

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[1] *Vide* Catalogue.

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[1] *Vide* Catalogue.

valvular lesions at this orifice. In certain cases of dilatation of the right ventricle, the auricular orifice becomes enlarged, the tricuspid valve not undergoing a corresponding increase in size, and the consequence is insufficiency of the valve. Tricuspid regurgitation, under these circumstances, plays an important part in the production of certain pathological effects and symptoms of cardiac disease, namely, jugular turgescence and pulsation, general dropsy, &c., which will be presently noticed. In post-mortem examinations, valvular insufficiency from this cause is liable to be overlooked unless attention be directed specially to the size of the orifice, which, in its normal condition, should not greatly exceed four inches in circumference. It was remarked first by John Hunter, in his treatise on the blood, that the tricuspid valves are not so well adapted to afford complete protection to the auricular orifice as the mitral valves, and hence he infers that it is less important for this orifice to be protected on the right than on the left side. Mr. Adams,[1] of Dublin, and afterward Dr. T. W. King,[2] of London, advocated the opinion that the tricuspid valves are disposed with special reference to regurgitation, and that an important function is to permit a retrograde current through the auricular orifice when the right ventricle becomes over-distended. Dr. King calls this the "safety-valve function." Experiments show that, after death, the tricuspid valves are insufficient when the right ventricle is distended with liquid. Injection into the ventricle causes a tricuspid regurgitant current; whereas, the injection of liquid into the left ventricle shows the mitral valves, under the same circumstances, to be sufficient to protect against regurgitation. Hence, in life, when the right ventricle is distended, and especially when to distension is added morbid dilatation, more or less regurgitation doubtless takes place at the commencement of the ventricular systole. But the different segments of the valve must quickly be brought into apposition during the systolic contraction of the ventricle, and further regurgitation prevented, provided the valve be sound and the auricular orifice not enlarged.

Obstruction of the coronary arteries may be noticed in connection with the pathological effects of valvular lesions of the heart. Obstruction of one or both of these arteries may arise from encroachment of masses of fibrinous or calcareous deposit upon their mouths, or an extension of these deposits into the vessels themselves. The supply of arterial blood to the substance of the heart is diminished in proportion to the contraction of the calibre of the vessels or of their openings into the aorta. It is reasonable to infer that enfeebled muscular action must be the immediate result, and impaired nutrition, involving softening and leading to dilatation, may follow. According to the observations of Quain,[1] obstruction of the coronary arteries is found in a pretty large proportion of the cases of fatty degeneration. He observed this complication in twenty-five of eighty cases. It is probably owing to the absence of free communication by anastomoses of the branches of the two coronary arteries that the obstruction of one of these arteries may give rise to important pathological effects. Atrophy of the muscular substance of the heart has been observed in connection with extreme obstruction from the atheromatous or calcareous deposit within these arteries. Formerly the paroxysms of severe pain which occur during the progress of some cases of organic disease of the heart, constituting the affection known as *angina pectoris*, were attributed to coronary obstruction; but clinical observation has abundantly shown this association to be by no means constant.

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[1] Dublin Hospital Reports, vol. iv.

[2] Essay on "The Safety-valve Function in the Right Ventricle of the Human Heart," by T. W. King, Guy's Hospital Reports, vol. ii.

As symptoms referable directly to the heart may be noticed, pain, palpitation, abnormal changes of the pulse, turgescence of the veins, and venous pulsation.

Pain.—Exclusive of paroxysms of suffering, frequently extremely severe, constituting the affection, superadded to certain cases of organic disease of the heart, known as *angina pectoris* (an affection to be considered hereafter), pain is not a prominent symptom of valvular lesions. In the majority of cases, this symptom is, in fact, wanting. Absence of pain is the rule, but occasionally patients complain of painful sensations referred to the præcordia. A sense of constriction, uneasiness, or undefinable distress, is oftener met with than actual pain. These sensations are not distinctive of organic disease; they are quite as likely to occur in connection with merely functional disorder of the heart. When present, they do not indicate the particular seat of the lesions, but it is undoubtedly true that they occur more rarely in connection with mitral than with aortic lesions. This statement will be found to hold good equally with respect to paroxysms of angina pectoris. When the existence of valvular lesions is determined by means of physical signs presently to be considered, the absence of pain, and, it may be added, tenderness or soreness of the præcordia, is of some weight in determining that the affection of the valves does not involve existing inflammation or endocarditis. But, as will be seen hereafter, pain, tenderness, or soreness are by no means always present when endocardial inflammation exists, so that absence of these symptoms is not proof against the existence of endocarditis.

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[1] Med.-Chir. Trans., *vide* Bellingham, op. cit.

Palpitation.—A person in health is not conscious of the action of the heart except when it is excited by exercise, mental emotion, or some other transient cause. The abnormal power of this action in some cases of organic disease, however, renders it perceptible to the patient. It may force itself on his attention, and occasion annoyance or suffering, the action being, in some cases, simply more or less intense, and perhaps, at the same time, irregular or intermitting. The consciousness of an undue force of impulsion is by no means a constant symptom of valvular affections. It does not occur till the heart becomes enlarged as a result of these affections. It is, in fact, due, not directly to the lesions of the valves or orifices, but to the hypertrophy to which they have given rise. When the patient complains of the beating of the heart, the impulse is found, on applying the hand over the præcordia, to be abnormally forcible. Portions of the dress or of the body may be visibly moved by the violence of the action. But the patient often does not complain of this symptom, and does not even notice it, when to the observer it is strongly marked. It is not unusual for patients to say that they have never experienced palpitation, when the action of the heart is perceived by the hand, applied over the præcordia, to be extremely violent and irregular. The explanation of this is, the abnormal force having been developed very gradually, the mind has become habituated to it, and is unconscious of it, at least unless the attention be directed to it. Palpitation, therefore, may be present as an objective, when it is wanting as a subjective, symptom. Hence, also, the inconvenience which it occasions does not always correspond with the degree in which it actually exists. Other things being equal, the increased violence of the heart's action is proportionate to the amount of hypertrophy, and especially hypertrophy of the left ventricle.

The consciousness of undue violence or of irregularity of the heart's action, exclusive of other circumstances, is not significant of organic disease. On the contrary, if the patient complain of this as a prominent symptom, the presumption is that organic disease does not exist. Palpitation from functional disorder always occasions great uneasiness, and generally intense anxiety and alarm. It is quite otherwise with palpitation incident to organic disease. It is surprising how insensible patients frequently are to the excessive force and great irregularity of the action of the heart, when due to structural affections, and how indifferent they often are when they are conscious of palpitation. The contrast, in this respect, between cases of organic disease and those of merely functional disorder is very striking. Other points serve to distinguish functional palpitation from that due to organic disease. The latter is less violent, but, to a greater or less extent, constant, while the former occurs in paroxysms, in the intervals of which the heart is tranquil. Palpitation from organic disease, increased beyond its habitual amount, is occasioned generally by some obvious cause, and more especially by muscular exercise. Functional palpitation occurs often when it cannot be traced to any exciting cause, and is more likely to occur when the patient is at rest than when engaged in active exertion. The former takes place more frequently in the daytime, the latter during the night. These and other points pertaining to the differential diagnosis will be considered more fully in treating of functional disorder of the heart.

As regards the relations of palpitation to the different valvular lesions, undue violence of the heart's action occurs more frequently, and, as a rule, is more marked, in cases of aortic, than in cases of mitral, lesions. This is owing to the fact that the former, more than the latter, tend to give rise to hypertrophy of the left ventricle. The violence of the impulse, of course, depends on the amount of enlargement by hypertrophy rather than by dilatation, and on the activity of the systolic contractions. The patient is more likely to perceive and suffer from the violence of the heart's action if the hypertrophy have been developed rapidly, than if its progress have been very gradual. The irregularity, of which the patient may, or may not, be conscious, depends, in a great measure at least, on the variations in the quantity of blood delivered to the cavities, in consequence of the interruption to the currents by the obstructive or regurgitant lesions. Irregularity of action due to these causes will be considered in connection with the pulse.

Pulse.—The characters of the pulse are often of considerable assistance in determining the situation, nature, and extent of lesions affecting the valves and orifices of the heart. The abnormal variations, which are important to be considered with reference to the points just mentioned, are its size, its strength as compared with the heart's impulse, its rhythm, the equality or inequality of successive beats, its quickness or slowness, the duration of the movement of the artery under the finger, &c. The frequency of the pulse, although important as representing the general condition of the circulation and the state of the vital forces, has no special significance as regards valvular disease.

In mitral lesions attended by regurgitation, the size and strength of the pulse are diminished in proportion to the quantity of blood driven backward, by the systolic contraction of the left ventricle, into the left auricle. If a small quantity only regurgitate, the pulse may still retain considerable volume and force; but if the amount of regurgitation be large, the pulse is notably weak and small. The weakness and smallness of the pulse are in contrast with the impulse of the heart, as felt by the hand applied over the præcordia, provided the left ventricle be hypertrophied and the action of the heart vigorous. The pulse may be regular, but often, in an advanced stage of the affection, its rhythm is disturbed; it becomes irregular or intermitting. Inequality of the pulse, that is, variation of successive beats as respects size, force, &c., is frequently observed, although it is less characteristic of mitral regurgitant lesions than of those attended by obstruction. The frequency of the pulse in this, as in the other varieties of valvular lesions, depends on the vital condition of the heart.

The graphical characters of the pulse as delineated by the instrument, devised by Marey, called the sphygmograph, present diversities by which the different valvular lesions are to some extent distinguished from each other. Referring the reader to

Marey's treatise,[1] or to works on physiology, for an account of the sphygmograph, of the normal characters belonging to the visible form of the pulse which this instrument delineates, and of the significance which Marey attaches to these characters, I shall limit myself to a brief statement of the different morbid characters distinguishing, severally, the different valvular lesions, together with typical illustrations copied from Marey's treatise.

As regards the graphical characters which are distinctive of lesions giving rise to mitral insufficiency, the "line of ascent" is short in proportion to the amount of regurgitation. In a series of the "forms" representing successive pulsations, this line presents a notable disparity in length, the disparity being in proportion to the unequal quantities of blood sent into the aorta with the successive systoles of the left ventricle. The line of ascent usually is vertical, showing deficiency of arterial tension. The "line of descent" is oblique in proportion to the slowness of the passage of blood from the arterial to the venous system; and it frequently also shows oscillations of the arteries, or dicrotism.[2]

Mitral obstructive lesions equally, but in a different manner, involve diminution of the size and strength of the pulse. In cases of mitral regurgitation, the pulse is rendered small and weak by the deduction of the blood which regurgitates from the quantity which would otherwise be propelled into the aorta with each systole. In cases of mitral obstruction, the blood not passing from the left auricle with sufficient freedom, the left

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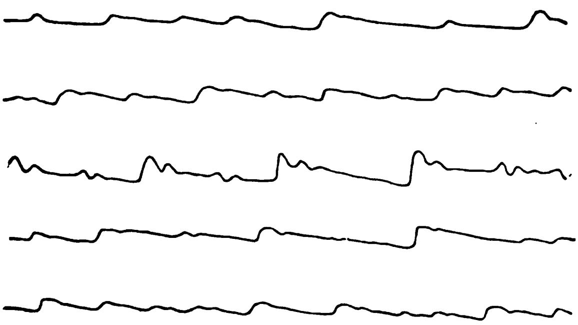
[1] Physiologie Médicale de la Circulation du Sang, &c., par le Docteur E. D. Marey, Paris, 1863.

[2] The following are typical illustrations of the characters denoting mitral lesions with notable insufficiency: —



ventricle fails to receive the quantity which should be propelled into the aorta. In the one case, the left ventricle is abundantly supplied, but it is not capable of conveying all its contents into the arterial system in consequence of the insufficiency of the mitral valves; in the other case, the supply to the left ventricle is deficient, and the blood accumulates in the left auricle and pulmonary vessels. In both cases the effect is the same so far as regards the lessened quantity of blood propelled into the aorta, and hence in both alike there occurs abnormal diminution of the size and strength of the pulse. Mitral contraction, when extreme, renders the pulse not only small and weak, but often irregular, intermitting, and unequal. The latter variations are observed especially when the pulse is at the same time frequent. The inequality depends on the varying quantity of blood which passes from the auricle to the ventricle between the successive systolic contractions of the ventricles. When the orifice is much obstructed, various circumstances prevent an equal supply of blood to the ventricle prior to the ventricular systole, and in proportion to the quantity of blood propelled into the aorta, other things being equal, will be the size and force of the pulse. When, from any cause, the supply of blood preceding the ventricular systole is less than usual, the pulse, which represents the systole of the left ventricle, is unusually small and weak. Under these circumstances the heart acts with irregularity. A greater deficiency of blood than usual causes the ventricle to contract for several beats with more frequency, as if to compensate by the number of systolic movements for the deficient supply of blood; hence, it is not unusual after several regular beats of the pulse, having a certain volume and strength, for a series of rapid beats to ensue, which are notably small and weak. As remarked by Dr. Adams, "it appears as if there were two pulses, one slow and deliberate for two or three beats, succeeded by three or four rapid and indistinct pulsations."[1] Intermittency of the pulse may represent intermittency of the heart's action, but it is sometimes observed when there is not a corresponding interruption in the heart's action. The apex-beats may be felt by the hand over the præcordia to take place in regular succession, while the pulse is found to intermit more or less frequently. This occurs in some cases of mitral obstruction, the explanation being that the quantity of blood delivered from the auricle to the ventricle, through the contracted orifice, is at times insufficient for the wave through the remote arteries to be perceived by the touch. The beats which, under these circumstances, are lost at the radial artery, may be distinguishable at an artery larger in size and nearer the heart, namely, the carotid. Intermittency of the pulse, it must be recollected, is a peculiarity of the circulation in some persons in health. An intermission, or the loss of a beat, occurs more or less frequently, the person not being conscious of its occurrence. It is not, therefore, intrinsically a symptom of disease. It is a curious fact that in persons who present this idiosyncrasy the pulse ceases to be intermittent in disease attended by febrile movement. A reappearance of the intermissions, under these circumstances, is evidence of the return of health. Weakness, smallness, and irregularity, as well as intermittency and even inequality, it is to be borne in mind, are not distinctive of mitral or other valvular lesions. All these characters of the pulse may occur in cases of enlargement or fatty degeneration not associated with lesions of the valves or orifices. All may occur, moreover, in merely functional disorder of the heart. A distinguishing point pertaining to the latter is, they occur only during paroxysms of palpitation presenting the distinctive features of palpitation from nervous disorder, whereas, occurring in connection with valvular lesions, they are either constant or frequently recurring, and unattended by the features which distinguish functional palpitation. It is also to be borne in mind that in cases of mitral obstruction, provided the contraction of the orifice be not great, the pulse may present sufficient size and strength, and it may be regular and equal. Obstructive lesions at the mitral orifice are less likely to lead to dilatation of the left ventricle than mitral regurgitant lesions ; hence, the latter, more than the former, occasion feebleness and irregularity of the pulse dependent on weakness of the ventricular walls superadded to the valvular lesions. The amount of either obstruction or regurgitation is represented by the smallness and weakness of the pulse, the more these characters are in contrast with the strength of the heart's impulse or felt by the hand in the præcordia. It will be recollected that mitral obstructive and regurgitant lesions are frequently combined. The symptomatic phenomena referable to the pulse will, of course, be more marked in such cases.

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[1] Dublin Hospital Reports, vol. iv. From Bellingham, op. cit.

The graphical characters of the pulse in a few cases of mitral obstructive lesions observed by Marey, show considerable regularity, and the visible forms are distinguished chiefly by a short line of ascent denoting the diminished quantity of blood propelled into the aorta by the ventricular systole. The application of the sphygmograph to a larger number of cases would doubtless have furnished illustrations, in addition, of irregularity and inequality, for the pulse undoubtedly may have these characters, as determined by the touch, when obstruction exists without regurgitation. My recorded cases furnish proof of this fact.[1]

Aortic lesions giving rise to obstruction are not characterized by a pulse weakened in proportion to the diminshed quantity of blood propelled from the left ventricle. Clinical observation shows that even when the contraction of the orifice is great, the pulse may retain considerable force. In an instance in which the orifice was reduced to the size of a quill, the pulse was neither small nor weak in a marked degree. This fact, which at first view may seem inconsistent, is intelligible when it is considered that a primary effect of aortic obstruction is hypertrophy of the left ventricle. The increased muscular power of this ventricle thus in a measure compensates for the reduction in size of the aortic orifice; and, as an additional mode of compensation, under these circumstances, the systolic contractions are apt to be prolonged, the blood, as stated by Blakiston, being "gradually squeezed through the contracted orifice." Moreover, the arteries are not unfilled to the same extent as in cases of great mitral obstruction and regurgitation, and the momentum communicated by the hypertrophied ventricle to the column of blood contained in the arteries may be sufficient to produce a pretty strong pulsation of the arterial trunks, even when obstructive aortic lesions exist to a considerable extent. Nevertheless, in cases of extreme contraction, the obstruction is too great to

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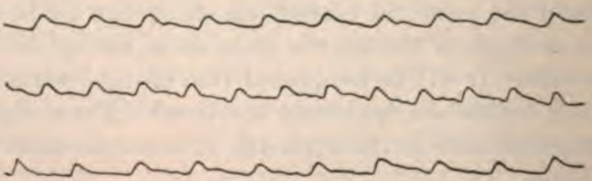
[1] The following are typical illustrations of mitral obstruction, as denoted by a presystolic murmur, from Marey's work:—



admit of compensation, and the pulse, under these circumstances, is small and weak. In cases of aortic obstruction, when great enlargement of the heart has ensued, and especially when the muscular power of the organ is much diminished by either dilatation or fatty degeneration, the pulse may become irregular, intermitting, and unequal. These deviations occur alike in aortic and mitral lesions at an advanced period of the disease. They occur, however, less frequently, at a later period, and in a less marked degree, in cases of aortic obstruction than in cases of either mitral obstruction or regurgitation. Irregularity and inequality are thus, in some measure, diagnostic of lesions affecting the mitral orifice as contrasted with those affecting the aortic orifice; but it is to be borne in mind that they occur in cases of dilatation, fatty degeneration, &c., uncomplicated with any affection of the valves or orifices. When the amount of aortic obstruction is sufficient to diminish, more or less, the size and force of the pulse, the impulse felt in the præcordia may be abnormally strong, owing to hypertrophy of the left ventricle. It is especially in cases of this description that a marked contrast between the pulse and the heart's impulse is observed. Slowness of the pulse, that is, the gradual expansion of the artery, as distinguished from quickness (not frequency) denotes the prolonged systole of the left ventricle, and is distinctive of aortic obstruction. Absence of the inequality of successive pulses is another negative differential point.

The graphical characters of the pulse, in cases of aortic obstruction, when the action of the heart has not been rendered weak, irregular, or intermitting by dilatation or fatty degeneration, are those which denote slowness without diminution of the power of the left ventricle. The obliquity and the length of the line of ascent are the characters which are distinctive of aortic obstruction, as contrasted with the characters of mitral lesions.[1]

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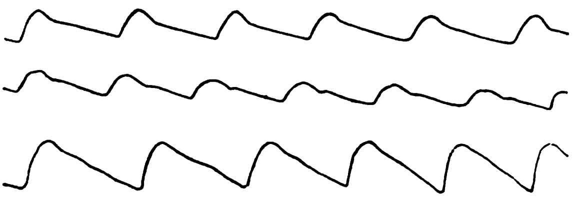
[1] The following are typical illustrations of aortic obstruction from Marey's work :—



Aortic lesions giving rise to regurgitation, if the regurgitant current be considerable, are characterized by a pulse which is in some measure diagnostic. When the aortic valves are sufficient, the column of blood contained in the arteries is supported by them, after the systolic contraction of the ventricle, and the elastic recoil of the arterial coats contributes in propelling the blood in its onward current. But if the valves be insufficient, the column of blood being incompletely supported after the ventricular systole, a quantity, greater or less, according to the extent of the insufficiency of the valves, flows backward into the ventricle, and the recoil of the arterial coats acts alike in producing an onward and a regurgitant current, so that when the contraction of the left ventricle takes place, the blood propelled into the aorta meets a regurgitant, instead of overtaking an onward, current. Clinical observation shows that under these circumstances, as was first pointed out by Corrigan, of Dublin, the pulse is notably quick and short, that is to say *jerking*. The artery strikes the finger suddenly, seemingly with considerable force, and appears instantly to recede. This has also been called a "collapsing pulse." It occurs, as a rule, in cases of aortic lesions with considerable insufficiency, and the jerking or collapsing feature is usually strongly marked. It is not a symptom of aortic obstruction, and inasmuch as the physical signs enable the diagnostician to determine the existence of lesions affecting the valves or orifice of the aorta, it is a symptom of importance as indicating that the lesions here situated are of a kind to permit free regurgitation.

The graphical characters of the pulse are highly distinctive of aortic regurgitation. The characters consist of a vertical line of ascent, representing the quickness of the pulse. The line of descent is at first nearly vertical, representing the rapid loss of arterial tension. Oscillation of the arteries during the ventricular diastole, constituting dicrotism, is shown by the irregularities of this line. The approximation of the line of descent at its commencement, to a vertical line renders the summit of the form of the pulse sharp and pointed.[1]

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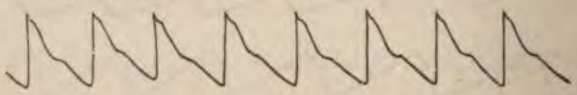
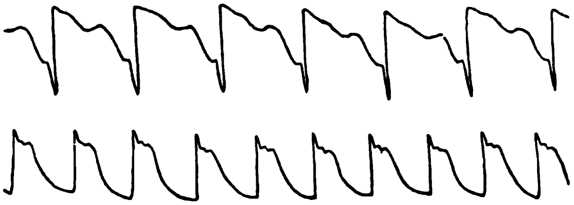
[1] The following are typical illustrations of the pulse denoting aortic regurgitation from Marey's work:—

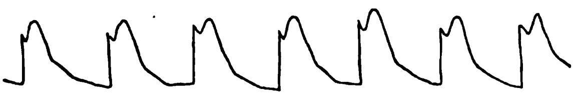


Visible pulsation of arterial trunks superficially situated, such as the subclavian, carotid, temporal, brachial, radial, &c., is a symptom characteristic of aortic regurgitation. If the insufficiency of the valves be great, an effect of the collision of the retrograde diastolic current and the onward systolic current in the vessels is to cause the latter, as it were, to be "suddenly thrown from their bed, bounding up under the skin." The visible pulsation is due, not alone to the diastolic movements of the coats of the vessels, but to the locomotion of the arteries. They "sometimes appear like worms under the skin, wriggling into tortuous lines at each pulse."[1] The connection of this symptom with aortic regurgitation, was first pointed out by Corrigan.[2] It is usually present whenever considerable regurgitation takes place, and it is often strongly marked. Taken in conjunction with the signs which establish the diagnosis of aortic lesions, this concurs with the jerking pulse in signifying that the lesions are of a nature to render the valve considerably insufficient.

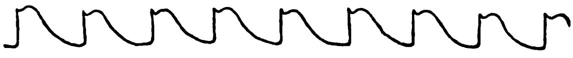
Another symptom pertaining to the pulse is significant of



The form of the pulse in cases of aortic obstruction and regurgitation combined is characterized by a line of ascent vertical until near the summit, and here having a notch from which the ending of the line is oblique. The following is a typical illustration :—



If, as is frequently the case, with aortic insufficiency the aorta be calcareous, so that the elasticity of its walls is much impaired, the force of the systole and the arterial tension are for an instant balanced, and the result is a horizontal line at the summit between the line of ascent and the line of descent. The following is a type of the visible form of the pulse under these circumstances :—



[1] Dr. Williams. Bellingham, op. cit.

[2] Edinb. Med. and Surg. Jour., vol. xxvii, 1832.

aortic regurgitation, namely, prolongation of the interval between the pulsation of the radial artery and the heart's impulse. The interval, as stated by Henderson, is sometimes so much lengthened "that the heart and the radial artery seem to beat with a distinct alternation." The rationale of this symptom is intelligible, in view of the conflicting currents within the large vessels which have been referred to in connection with the production of a jerking pulse and visible pulsation of the arteries. Some observers have been led to doubt the occurrence of this symptom. That it characterizes certain cases in which the regurgitation is excessive, is not to be denied. In a case under my observation of mitral and aortic regurgitation, with great hypertrophic enlargement, the interval between the apex-beat and the beat of the radial artery was so much longer than that between the first and second sound of the heart, that the radial pulse was, in fact, in closer relation to the diastole than to the systole of the left ventricle. The interval between the apex-beat and the pulsation of the carotid artery was less, being about the same as exists normally between the apex-beat and the radial pulse. The visible movements of the arteries, and the jerking character of the pulse were strongly marked in this case. The *sphygmoscope*, devised by Dr. Scott Alison, of London, is well adapted to illustrate the relative occurrence of the apex-beat and the arterial pulse, in different situations.

Turgescence of the Veins and Venous Pulsation.—Abnormal fulness of the veins occurs whenever there is an obstacle to the free entrance of blood into the right auricle. An obstacle exists when the right auricle is already full, or distended with blood. Various abnormal conditions involve this result. The most direct and efficient causative condition is contraction of the tricuspid orifice. But this is rare; so much so, that the probabilities of its existence in a given case of distension of the right auricle are hardly sufficient for it to be taken into account. Tricuspid regurgitation is another condition leading to the result. This is much less infrequent than tricuspid contraction. The explanation of distension of the right auricle when, from insufficiency of the tricuspid valve, a portion of the contents of the right ventricle is driven backward with each systole, is obvious. Dilatation of the auricle follows sooner or later. Lesions affecting the valve or orifice of the pulmonary artery also induce distension and dilatation of the right auricle, exerting an effect primarily on the right ventricle. Contraction and insufficiency here situated are as infrequent as tricuspid obstruction, if we except congenital lesions. In the affections developed after birth, therefore, these are to be excluded as probable conditions giving rise to venous turgescence. Distension and dilatation of the auricle, however, occur irrespective of lesions affecting either the tricuspid or pulmonic orifice. They occur, as has been seen, especially in connection with mitral lesions which involve obstruction or regurgitation, either separately or combined. The right ventricle in these cases first becomes over-distended and enlarged, and distension and dilatation of the right auricle follow. Turgescence of the superficial veins is therefore observed, not alone in cases of tricuspid and pulmonic, but also in cases of mitral lesions when the latter have led to enlargement of the right side of the heart. It is equally a symptom of the latter when not induced by valvular lesions. Obstruction to the pulmonary circulation from emphysema of the lungs occasions an undue accumulation of blood within the right ventricle and auricle, leading after a time to enlargement and a consequent obstacle to the free transmission of blood from the systemic veins. Finally, pressure on the vena cava by an intra-thoracic tumor produces obstruction and venous turgescence. Thus, marked fulness of the veins of the head and neck is observed in some cases of aneurism of the arch of the aorta. As a symptom, then, this is not distinctive in itself of cardiac disease, nor, when it proceeds from the latter, does it point to the seat, or even denote the existence, of lesions of the valves or orifices. Exclusive of the cases in which it is an immediate effect of extra-cardiac obstruction, it simply shows that the right auricle is either dilated or over-distended.

Venous turgescence may be apparent wherever the superficial veins are visible, but it is usually most conspicuous, on the neck, in the jugulars and the venous branches communicating with them. In some instances of extreme turgescence the vessels present a varicose appearance. These veins may be habitually full and dilated, as they are seen to be temporarily during prolonged expiratory efforts in singing, playing on wind instruments, straining, and in paroxysms of spasmodic cough. If the obstruction at the right auricle be considerable, when pressure is made on a vein high on the neck, the vessel remains distended below the point of pressure, and may be refilled after the contents of the vessel have been pressed backward by the finger, showing not only a resistance to the gravitation of the blood, but a reflux current.

This symptom, taken in connection with the physical signs which establish the nature and seat of organic lesions of the heart, possesses considerable value. If it proceed from lesions situated at the tricuspid or the pulmonic orifice, the concurrent signs will show the existence of these lesions, and the degree of venous turgescence will be, to some extent, an index of the extent to which they occasion obstruction. If, on the other hand, it proceed from mitral lesions, the signs enable us to localize these, and the turgescence is then evidence, and its degree in some measure a criterion, of the effect which they have produced on the right side of the heart. It shows that the mitral lesions involve an amount of obstruction which will lead to dilatation of the right auricle if it have not already taken place. It shows also an effect on the systemic circulation which involves a liability to other effects dependent on it, namely, dropsy and extravasation of blood or hemorrhage. In these points of view it is a symptom which deserves attention in the examination of patients affected with cardiac disease. It is, of course, understood that the value of this symptom, in its relation to disease of the heart, depends on the absence of extra-cardiac conditions, such as emphysema or aneurism, which may give rise to it. Careful examination will enable the diagnostician either to exclude these conditions or to ascertain their presence in individual cases.

Venous pulsation is a diastolic movement of the veins which is visible and sometimes even appreciable by the touch. The movement is due to a retrograde current or impulse communicated to the blood contained in the veins. It is to be distinguished from the movements occasioned by respiration, with which every one is familiar, and also from those communicated by subjacent arteries. With the latter it is liable to be confounded, unless care be taken to avoid this error. The error may be avoided by making moderate pressure over the veins at the lower part of the neck. Pressure here, not sufficient to stop the circulation in the arteries, will arrest pulsation of the veins. The movements due to respiration may be arrested by causing the patient to suspend breathing for a few seconds. Pulsation is rarely observed elsewhere than in the veins of the neck. It is often limited to the veins just above the clavicles. It may be limited to one side of the neck, and, when this is the case, it is usually observed on the right side. In some instances, however, the pulsation extends to the superficial veins at remote parts of the body. It has been observed even on the dorsal surface of the hands.[1] Pulsation is often, but not always, accompanied by turgescence of the veins, and is especially marked at the end of the act of expiration, when the fulness is greatest. It may also be increased by pressure on the vein above the point where it is observed. It varies in degree or force between a very gentle undulatory movement perceptible to the eye, and a movement which is not only seen, but communicates a sensation to the finger sufficiently distinct but never strong.

As a symptom of cardiac disease, venous pulsation was first described by an Italian author, Lancisi, who ascribed it to dilatation of the right ventricle. The phenomenon, however, had been previously noticed by another Italian writer, Testa.[2] The inquiry which first arises is, Does it invariably denote disease? Dr. J. W. King, in connection with the subject of the safety-valve function of the tricuspid valves, adduces cases to show that it occurs independently of any organic disease of the heart; and Dr. Francis Sibson states that "a slight systolic pulsation is visible, below the sterno-cleido, in the superficial jugular, in thirty-nine persons out of forty when they lie down."[3] Assuming that a venous pulsation may exist without being abnormal, the supposition, which has been entertained, that it may be produced by the impulse communicated by the contraction of the left ventricle being transmitted through the capillary vessels (*vis à tergo*), is not tenable. It must be due, in health as in disease, to a reflux movement, and hence it follows, assuming its occurrence to be synchronous with the ventricular systole, either that an amount of tricuspid regurgitation, irrespective of disease, sometimes occurs sufficient to give rise to a retrograde current extending to the cervical veins, or, as contended by Hope, the expansion of the curtains forming the tricuspid valves during the ventricular systole suffices to impart to the blood contained in the right auricle a momentum which extends to the column of blood contained within the veins for a certain distance. The latter view is not improbable, and both explanations are perhaps admissible. But it is only when the pulsation is slight and circumscribed that there is room for doubt as to its being a symptom of disease. If it be marked or extensive, it is, in a certain proportion of cases, evidence of tricuspid regurgitation. As a symptom of tricuspid regurgitation, it is more valuable in a positive than in a negative point of view: that is, while, if present, it denotes tricuspid regurgitation, its absence is not proof of the non-occurrence of this regurgitation. An important element in its production is involved, in addition to insufficiency of the tricuspid valves, namely, increased, or at least undiminished, muscular power of the right ventricle. If the contraction of this ventricle be feeble, from dilatation or fatty degeneration, the regurgitant current is not strong enough to extend much, if at all, beyond the auricle into the veins. On the other hand, the force of the regurgitation and the reflux into the veins, other things being equal, will be commensurate with the power with which the right ventricle contracts. Hence, it is obvious that the symptom under consideration is not only valuable as evidence of tricuspid regurgitation, but also as constituting, in some measure, an index of the energy of the systole of the right ventricle. The conditions most favorable for the production of the venous pulse are free tricuspid regurgitation and hypertrophy of the right ventricle. These combined conditions are present in connection, not only with valvular lesions seated in the right side of the heart, but, as has been seen, with lesions affecting the mitral orifice in a certain proportion of cases. It is easy to understand that hypertrophy of the right ventricle, without tricuspid regurgitation, may exaggerate the pulsatory movements of the cervical veins which are often observed in a slight degree in health; but it is probably correct to say that hypertrophy of the right ventricle alone, that is, without tricuspid regurgitation, does not give rise to this symptom in a marked degree.

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[1] A Case of Pulsation in the Veins of the Upper Extremities. By Charles Benson, M. D. Dublin Journal of Medical Science, vol. viii, series No. 1, 1836. *Vide* Stokes. op. cit., Am. ed., p. 219.

[2] *Vide* Stokes, op. cit., Am. ed., p. 214, note.

[3] Medical Anatomy. London. Fasciculus, No. 1.

Venous pulsation is to be explained in the manner just stated in a certain proportion of the cases in which it is observed. In some cases, however, it is otherwise produced. The systolic contraction of the right auricle may cause a movement of the blood in a retrograde direction sufficiently to give rise to this symptom. Experimental observations show that the auricular systole precedes, by a very brief interval, the ventricular, the former being, as it were, continued into the latter. A venous pulse due to auricular contraction should therefore precede slightly the arterial pulse or apex-beat of the heart. This point suffices for the discrimination between an auricular and a ventricular venous pulse.

Placing the finger over the apex of the heart, the apex-beat, if it be felt, will be found to follow the venous pulse in the neck, provided the latter be produced by the contraction of the auricle; whereas, if the venous pulse be due to tricuspid regurgitation, it will be either synchronous with or lagging a little behind the apex-beat. Not infrequently, however, an apex-beat is not appreciable to the touch; then another, and generally a more convenient, mode of determining whether a venous pulse be auricular or ventricular, is to observe if it precede or occur synchronously with the arterial pulse of the neck. This can be done without difficulty. With the finger placed over the carotid, and the eye fixed upon a pulsating vein, it is easy to determine whether the venous pulse precedes or occurs simultaneously with the beating of the artery. Frequently this can be determined by the eye alone, the carotid and the venous pulse being at the same time visible. A comparison of the venous with the radial pulse will not suffice to determine whether the venous pulse be auricular or ventricular, as there is an appreciable interval of time between the radial pulse and the apex-beat of the heart, whereas the carotid pulse and the apex-beat are synchronous.

In some cases the auricular and the ventricular pulse are combined. In these cases there is a pulsation of the veins synchronously with, and another pulsation just preceding, the apex-beat or the carotid pulse. This I have observed in a considerable number of cases.

An auricular venous pulse is probably never appreciable elsewhere than in the veins of the neck. Indeed, it is extremely rare for the ventricular pulse to be apparent elsewhere. I have noted in some cases the occurrence of an auricular and a ventricular pulse only during the act of inspiration. A venous pulse in the neck may be apparent when the patient is recumbent, and not in a sitting or standing posture.

Finally, the caution may be repeated, not to mistake an arterial pulsation beneath a cervical vein for a venous pulse. A little pressure on the vein below the point where a venous pulse is apparent, will arrest it; whereas, if the pulsation be arterial, it will continue when the pressure is made. This test is always available.

SYMPTOMS AND PATHOLOGICAL EFFECTS REFERABLE TO THE CIRCULATION.

The symptoms just considered, namely, the abnormal variations of the pulse, turgescence of the veins, and venous pulsation, relate to the circulation, but they are due immediately to the heart, and hence represent directly its morbid conditions. Other phenomena of disease relating to the circulation are developed as ulterior effects of valvular affections, involving intermediate conditions, and, therefore, representing indirectly cardiac lesions. Under the present head are embraced two important events incidental to the clinical history of affections of the valves and orifices, namely, dropsy and arterial obstruction from vegetations or masses of fibrin detached from the endocardial membrane and carried with the current of blood into the vessels. Important pathological events, in addition to these, relating to the circulation, will be more appropriately considered in connection with phenomena referable to different organs. Such are hemorrhage, apoplectic extravasation, &c.

Cardiac Dropsy.—Transudation into the subcutaneous areolar tissue, together with the pleural, the peritoneal and other serous cavities, in other words, general dropsy, occurs sooner or later in a large proportion of the cases of valvular affections in which a fatal termination does not take place in consequence of some intercurrent or incidental disease. General dropsy, when dependent on disease of the heart, is called cardiac dropsy. It may be due to other pathological conditions, and especially to disease of the kidney, when it is distinguished as renal dropsy. It appears first, as a rule, in the form of œdema of the feet and ankles, which gradually extends over the lower extremities. Œdema of the face follows, sometimes occurring nearly simultaneously with, and occasionally prior to, swelling of the feet. It may extend over the whole body, and it is then called anasarca. The lower extremities, in some cases, become enormously swollen. Erythema and occasionally gangrene result from the extreme distension of the integument. Blisters, ulcerations, and cracks are other consequences, giving exit to the transuded liquid which flows away in abundance. The surface, more especially the face, presenting at the same time more or less venous congestion, the general aspect in cardiac dropsy is somewhat characteristic. The face has a dark or dusky hue, forming a striking contrast to the pallid complexion which is usually marked in cases of dropsy from renal disease. Effusion into the different serous cavities takes place subsequently to the subcutaneous œdema. The different serous cavities are by no means equally liable to dropsical effusion. Peritoneal effusion is the most frequent, and next in frequency is effusion into the pleural cavities. It is, however, rarely, if ever, the case that either hydroperitoneum or hydrothorax occurs without the other; but in some cases the pleural effusion is much greater than that in the peritoneal cavity. Effusion within the pericardium, more or less, but rarely in great quantity, may be added to hydroperitoneum and hydrothorax; and there is in some cases a marked increase of the liquid within the arachnoid cavity and in the subarachnoid space. Effusion into the pulmonary air-cells, constituting œdema of the lungs, occurs not infrequently. This will be noticed in connection with the pathological effects referable to the pulmonary system.

The occurrence of dropsy has reference to the situation, nature, and degree of valvular lesions. Tricuspid contraction is the lesion which most directly and efficiently tends to give rise to this effect; but this lesion is exceedingly infrequent. Tricuspid regurgitation exerts a similar tendency, but in a much less degree. Dropsy by no means uniformly occurs in the cases in which turgescence of the veins and a ventricular venous pulsation show marked insufficiency of the tricuspid valves. Of lesions situated in the left side of the heart, mitral contraction is most likely to give rise to dropsy. Mitral regurgitant lesions come next in order, as regards this tendency. Aortic lesions, very much oftener than mitral, continue and terminate without this pathological effect; but they are not always exempt from it.

By what mechanism is the dropsy produced? So far as concerns the agency of the cardiac lesions, the intermediate morbid condition is passive congestion of the systemic veins. To understand the *modus operandi*, we have to inquire in what manner the different obstructive and regurgitant lesions induce this venous congestion. This is sufficiently intelligible as regards lesions seated at the tricuspid orifice. The relations of the systemic veins to the right side of the heart are such that the rationale of dropsy, as an effect of obstruction and regurgitation situated here, is at once evident. Dropsy follows mitral lesions in consequence of the distension and dilatation of the right ventricle and auricle to which these lesions give rise. Repletion of these cavities constitutes an obstruction which may induce sufficient congestion of the systemic veins to lead to serous transudation. And when dropsy is an effect of aortic lesions, it takes place only after dilatation of the left ventricle has rendered the mitral valves insufficient, and dilatation of the right side of the heart has ensued. Thus, so far as concerns the relation of dropsy to valvular lesions, they all alike produce this effect by obstruction at the termination of the systemic venous system, namely, the right auricle, tricuspid lesions, involving directly this obstruction, and lesions situated at the mitral or aortic orifice, leading indirectly to the same result.

From the foregoing remarks it follows that the occurrence of dropsy, other things being equal, in cases of mitral or aortic lesions, will depend, not immediately on the nature and extent of these lesions, but on conditions induced thereby which relate to the right side of the heart. When to distension of the right ventricle and auricle is added either dilatation or weakness of the right ventricle, the venous obstruction due to over-accumulation of blood is increased by the diminished ability of this ventricle to contract and expel its contents. In point of fact, dilatation of the right ventricle or weakness from fatty degeneration and other causes, precedes; in the great majority of cases, the occurrence of dropsy. Dropsy, therefore, is an event which usually belongs to an advanced period of organic disease, and it is frequently a precursor of a fatal termination. Enlargement of the right side of the heart, especially if accompanied by degeneration of structure or great muscular weakness, may induce dropsy when valvular lesions are not present. The occurrence of dropsy, thus, when aortic or mitral lesions are present, is evidence that the effects of these lesions on the right side of the heart, which have been considered under another head, have taken place.

The mechanism of dropsy occurring in connection with valvular affections, so far as at present considered, involves simply mechanical pressure. The serous or watery portion of the blood transudes through the coats of the vessels in consequence of their distension, in the same manner that œdema of an extremity is induced by the obliteration of an important venous trunk coming from it. Morbid conditions, in addition to those giving rise to venous obstruction, however, may concur in producing cardiac dropsy. The frequent concurrence of other causes is shown by the absence of dropsy in cases in which the conditions pertaining to the heart must necessarily have involved, for a long period, marked congestion of the systemic veins; not only aortic, but mitral lesions involving a great degree of regurgitation and contraction, may end, after a protracted duration, without having led to dropsy. It is shown, also, by the want of proportion, which all clinical observers must have noticed, between the amount of dropsical effusion and the degree of obstruction which the cardiac lesions involve. Not only are these lesions marked in cases in which dropsy has not occurred, but dropsy occurs in other cases in which the lesions are comparatively slight. Evidently, then, something more than mechanical pressure is concerned in the production of dropsy in a certain proportion of the cases of cardiac disease in which this pathological effect takes place. Clinical experience shows that in some instances a concurring morbid condition is disease of kidney. Disease of heart and the affections of the kidney constituting Bright's diseases, are occasionally found associated. The causes which induce dropsy in the latter affections, then co-operate with those relating to the heart; renal and cardiac dropsy are, in fact, combined. Anæmia or hydræmia, arising from various causes, may determine the occurrence of dropsy when the cardiac lesions of themselves would not have produced it. Transudation of the attenuated serum, it is well known, may take place as a result of this condition of the blood when neither cardiac nor renal disease exists. Walshe attaches importance to the impaired nutrition of the walls of the vessels from the strain incident to prolonged distension, as a subsidiary cause of transudation. These additional conditions are to be taken into account in explaining the production of dropsy in cases of cardiac disease; and they serve to explain the efficiency of therapeutical measures in some instances in which the cardiac lesions are such as to render the continuance of venous congestion inevitable.

Arterial obstruction by vegetations or fibrinous masses detached from the valves or orifices of the heart. Embolism.—As already remarked, in describing the morbid appearances which are presented in cases of lesions of the valves and orifices, vegetations, or fibrinous masses are frequently, in examinations after death, found to be so slightly adherent that it is reasonable to suppose the current of blood to be sufficient to detach them during life. They are carried onward with the current in the course of the circulation, until they reach an arterial trunk smaller than their own dimensions. Here they are arrested, and, becoming wedged in the vessel, they act as plugs, obstructing the passage of blood in the artery and its branches beyond the point at which they are lodged. They were called *emboli*, or migratory plugs, by Virchow, who appears to have been the first to make obstruction of the systemic arteries, as thus produced, a subject of scientific study.[1] Others have contributed the results of their researches, among whom Dr. William Senhouse Kirkes, of London, is to be especially mentioned.[2] Emboli, or plugs, may also be formed in the arteries and in the veins; and when formed in the veins they may be transported to the heart, and thence into the branches of the pulmonary artery. Emboli consisting of vegetations or fibrinous masses detached from the valves, do not embrace all which may be derived from the heart. Fibrinous clots formed under various pathological circumstances, also constitute emboli. The subject of embolism, therefore, is not limited to valvular lesions in its application, and it will be referred to hereafter, in treating of endocarditis and the formation of coagula within the heart.

Emboli from within the heart, in cases of chronic valvular lesions, are derived, in the vast majority of cases, from the left ventricle, since lesions affecting the valves of the right side of the heart are exceedingly infrequent. The obstructed arteries, therefore, belong to the systemic class, the emboli passing with the current of blood into the aorta and along the successive arterial trunks, until arrested in their progress by branches, the calibre of which is too small to permit their farther progress. The situation in which an embolus becomes fixed, will depend on its size and the direction which it happens to take. Owing to the large quantity of blood sent to the brain, it will be likely to take that direction, and produce obstruction of some one of the cerebral arteries. The embolus is most likely to be carried into the middle cerebral artery of the left hemisphere, owing to the fact that the current of the blood to this hemisphere is more direct than that on the right side, the left carotid springing from the arch of the aorta, whereas, the right carotid springs from the arteria innominata, and the latter forms an angle with the aorta. The plugging of a cerebral artery, by cutting off the supply of blood to a portion of the brain, may occasion sudden coma and hemiplegia, and the interruption of nutrition may lead to softening of the cerebral substance. These effects will be noticed in connection with pathological events referable to the nervous system. Or, the embolus may follow the current downwards through the descending aorta, and become lodged in some one of the secondary branches. Obstruction of the renal, splenic, iliac, femoral, and other arteries may be thus produced. Numbness, impaired muscular power of the lower extremities, loss of pulsation in the arteries accessible to the touch, and even gangrene, may result from obstruction of the iliac and femoral arteries.

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[1] *Vide* Brit. and For. Med.-Chir. Rev., July, 1857, p. 15. Virchow's earliest researches were in 1845.

[2] "On some of the Principal Effects resulting from the Detachment of Fibrinous Deposits from the Interior of the Heart and their mixture with the Circulating Blood," by William Senhouse Kirkes, M.D., Trans. Med.-Chir. Society of London, 1852.

The occurrence of arterial obstruction by emboli derived from the heart is rendered probable by the facts already stated, namely, the frequency with which loosely attached masses of variable size are observed on the valves in post-mortem examinations, and the occurrence of paralysis and other effects, in cases of valvular disease, which may be thus explained. But the proof rests on the identity of the emboli or plugs found in the arteries with vegetations or fibrinous masses existing at the same time on the valves. The fact of obstruction being ascertained, together with the existence of local changes (in the brain especially), which are due to this obstruction, the obstructing embolus may not only resemble in its gross appearances coexisting vegetations or masses of fibrin within the heart, but it has the same composition and formation as determined by microscopical examination.

Emboli derived from within the heart are sometimes calcareous. I have met with a case in which large calcareous masses had formed within the right ventricle, and a portion of the size of a pullet's egg had been detached, and become impacted in the left division of the pulmonary artery. This vessel was completely obstructed by the embolus, and the fractured surface showing the situation of its attachment within the ventricle was apparent. Pulmonary tuberculosis existed in this case, the tuberculous affection being greatest on the right side. A similar case, as regards plugging of a primary branch of the pulmonary artery by a fractured portion of a calcareous tumor in the right auricle, will be given in Chapter VI, under the heading, "Polypi of the Heart."

Other effects attributed to the removal of morbid products on the valves or orifices of the heart, and their transportation with the current of blood into the arteries, may be here alluded to. Instead of being detached in masses of greater or less size, they may be disintegrated and carried away in small particles. It is easy to conceive of a considerable quantity of the debris of fibrinous and calcareous deposits in this way accumulating within the vessels. This does not occasion obstruction of arterial trunks like plugs or emboli, but, accumulating in the minute or capillary vessels in certain organs, they may occasion local congestions and impaired nutrition. Other and more serious consequences have been imputed to the admixture of these particles with the blood. Typhoid symptoms, petechial eruptions, &c., occurring in the course of heart affections have been thus explained. This pathological view is, and must of necessity continue to be, hypothetical, since it is difficult, if not impossible, to demonstrate the presence of these particles, and their derivation from the heart. The doctrine has reference more to the recent products of inflammation deposited on the valves than to those of long standing which characterize chronic valvular lesions. The latter, from their consistency, are more likely to be detached in masses than to be disintegrated and carried away in fine particles, while soft lymph or fibrin is readily removed in the latter mode.

SYMPTOMS AND PATHOLOGICAL EFFECTS REFERABLE TO THE RESPIRATORY SYSTEM.

The phenomena referable to the lungs, irrespective of associated or intercurrent pulmonary affections, depend, for the most part, on vascular engorgement of these organs. Congestion of the lungs is an immediate result of an impediment to the free admission into the left auricle of blood from the pulmonary veins. An impediment exists whenever the left auricle is over-distended with blood; and over-distension of this auricle occurs as a consequence of any interruption of the blood-currents through the orifices of the left side of the heart. These effects are involved more directly and in a greater degree in mitral than in aortic lesions. Obstructive lesions at the mitral orifice especially give rise to pulmonary congestion. The phenomena dependent thereon are most marked, other things being equal, in cases characterized by extreme contraction of this orifice. Mitral regurgitation leads to pulmonary congestion and its dependent phenomena, but not so readily nor to the same extent as in cases of obstruction. The effects of regurgitation, however, are often added to those of contraction. Aortic lesions, obstructive and regurgitant, also, sooner or later, are followed by over-distension and dilatation of the left auricle and consequent congestion of the lungs. Dilatation of the left ventricle, however, precedes these effects, and the latter are produced more tardily than when the lesions are situated at the mitral orifice. In the rare instances of lesions occasioning obstruction at the tricuspid and the pulmonic orifice, the over-distension of the right auricle which ensues constitutes an impediment to the circulation in the systemic vessels, not extending to the pulmonary vascular system until the left ventricle becomes dilated. Engorgement of the lungs, therefore, is especially a pathological effect of lesions affecting the orifices at the left side of the heart, as congestion of the systemic veins is, in like manner, especially a pathological effect of an impediment existing in the right side. This statement applies to valvular lesions. It does not apply to another morbid condition which may contribute to pulmonary congestion, namely, weakness of the right ventricle from dilatation or fatty degeneration. The blood accumulates in the vessels of the lungs in consequence of not being propelled by this ventricle with sufficient power. Venous congestion is then due to deficiency in the *vis à tergo*. Dilatation of the right ventricle, which is an effect of the engorgement of the lungs incident especially to mitral lesions, thus tends to augment the difficulty of the circulation through the pulmonary circuit. An enfeebled condition of the right ventricle is, in itself, adequate to produce pulmonary congestion, as is seen in cases of fatty degeneration affecting this ventricle, but not to so great an extent as valvular lesions involving interruption of the blood-currents through the orifices in the left side of the heart. These remarks are alike applicable, *mutatis mutandis*, to weakness from dilatation or fatty degeneration of the left ventricle in connection with congestion of the systemic veins.

The engorgement of the lungs arising from valvular lesions gives rise to important pulmonary symptoms without any other superinduced affection of these organs. The most prominent of these symptoms are dyspnœa, cough, muco-serous expectoration, and hæmoptysis. Certain pulmonary affections appear in some cases to be dependent directly and exclusively on over-distension of the vessels, namely, extravasation of blood, or apoplexy of the lungs, and œdema. Other affections are incidental to valvular lesions, the state of congestion predisposing to them, or favoring their development. The existence of valvular lesions thus involves a liability to bronchitis, pneumonitis, pleurisy, and emphysema.

Dyspnœa is a symptom more or less prominent in the great majority of cases. If there be no superinduced or incidental affection of the lungs, the difficulty of breathing is proportionate to the amount of pulmonary congestion. It is a criterion of the extent to which the changes effected by respiration are compromised in consequence of the retarded flow of blood through the capillary vessels. It occurs earlier and is more marked in cases of mitral than aortic lesions, because the former tend more directly and in a greater degree to engorgement of the pulmonary vessels. In most cases of either mitral obstruction or regurgitation, dyspnœa is the first symptom which occasions inconvenience. The patient often complains of this symptom alone, or chiefly, for a considerable period. In cases of aortic lesions it occurs later, and is oftener preceded by palpitation or other symptoms referred by the patient to the heart. It is not uncommon to meet with examples of great contraction and insufficiency at the aortic orifice, accompanied by considerable enlargement of the left ventricle, when there had been little or no embarrassment of respiration. Instances are much less frequent of a similar amount of obstruction or regurgitation at the mitral orifice, which had not given rise to dyspnœa. When dependent on aortic lesions, this symptom is evidence of enlargement of the heart, since pulmonary engorgement does not occur until the left ventricle becomes dilated. When dependent on mitral lesions, dyspnœa may be experienced prior to much enlargement, the right ventricle becoming dilated or hypertrophied as an immediate result of the retarded circulation through the lungs. To the condition which the symptom represents, namely, pulmonary congestion, enlargement of the heart is thus antecedent in cases of aortic, and consecutive in cases of mitral lesions.

The intensity of dyspnœa varies greatly in the different cases of valvular affections in which this symptom is present, and in the same case at different periods. It consists, at first, of a slight deficiency of breath on exertion. This progressively increases until active exercise becomes insupportable. If the patient pursue an occupation which requires strong muscular movements, he finds it difficult, after a time, to continue them, and is at length compelled to give up labor. Cases frequently at this juncture first come under medical observation. The breathing may be sufficiently easy so long as quietude of the body is maintained, when the dyspnœa is marked on taking moderate exercise, even walking across the room. Habitual dyspnœa, in some instances, does not occur, or it takes place only during the latter part of life. Other cases are characterized by paroxysms of difficult breathing when not provoked by exercise, and by more or less difficulty which is constant. There is a notable difference in different cases as regards the sense of the want of breath, and the amount of suffering occasioned by the same apparent difficulty. The breathing is sometimes evidently labored when the patient makes no complaint, and says he experiences no inconvenience. This is probably owing, in part, to the symptom having been so gradually and imperceptibly developed that the mind becomes accustomed to it, and it is scarcely noticed so long as the habitual amount of difficulty only exists. The distress is not commensurate with the manifestations of difficulty, in other instances, because the perceptions are blunted by the circulaton of imperfectly oxygenated blood. In these instances more or less lividity of the prolabia and surface of the body is apparent. The suffering, however, is often great. The want of more breath is painfully felt, amounting sometimes to a sense of suffocation. The patient cannot lie down, but is obliged to keep the sitting posture, often bending forward and supporting himself by the hands locked below the knees, or resting upon some solid body. The accessory muscles are brought into play, to produce the greatest possible expansion of the chest. The countenance expresses great anxiety, and frequently the lips and face are livid. Dyspnœa having this intensity is distinguished as *orthopnœa*. Occurring in paroxysms, it constitutes the *cardiac asthma* of some authors.

These diversities as regards embarrassment of respiration show that this symptom is affected by a variety of circumstances. As an objective symptom, its intensity corresponds to the amount of pulmonary congestion, provided no other affection of the lungs be present. Subjectively, its intensity depends, in a great measure, on the rapidity or slowness with which the pulmonary congestion has ensued: that is, the suffering from dyspnœa, when the congestion is rapidly developed, is far greater than when it has been gradually induced. The striking difference among different persons in suceptibility to painful impressions serves also to explain the greater tolerance by some patients than by others of apparently an equal amount of difficulty of breathing. Affections of the pulmonary organs, superadded to congestion, contribute to increase the amount of dyspnœa. Emphysema, in connection with valvular lesions, in proportion to its extent, adds intensity to this symptom. This combination is not infrequent, and the cardiac affection is liable to be overlooked unless due attention be directed to an examination of the heart. Pleuritic effusion increases the dyspnœa by diminishing the volume of lung and limiting the range of thoracic expansion. Bronchitis produces the same effect, the supply of air to the cells being diminished by swelling of the bronchial mucous membrane and the accumulation of mucus within the tubes. Œdema compromises the breathing capacity of the lungs in proportion as the air-cells become filled with effused liquid. The coexistence of these or other pulmonary affections of course invalidates, to a greater or less extent, the significance of dyspnœa as representing the amount of pulmonary congestion due to valvular lesions. It is only when pulmonary complications are excluded that this symptom is to be considered as a criterion of the impediment to the pulmonary circulation which these lesions occasion. Spasm of the muscular fibres of the bronchial mucous membrane—in other words, true asthma—may occur in connection with valvular lesions. This explains, in some instances, the occurrence of the paroxysms of dyspnœa or orthopnœa which constitute cardiac asthma. Various circumstances, however, which occasion, temporarily, a considerable increase of the pulmonary congestion, may give rise to these paroxysms.

Cough and expectoration are usually present when valvular lesions have induced considerable pulmonary engorgement. The congested state of the bronchial mucous membrane leads to an abnormal secretion of mucus, and to transudation through the coats of the vessels into the tubes. The expectoration is muco-serous in its character. Varying in amount in different cases, it may be considerable without involving inflammation of the membrane. The affection, under these circumstances, is bronchorrhœa. But bronchitis is apt to become developed, giving rise to more cough, with an expectoration of modified mucus and muco-purulent sputa. The prominence of the cough and the characters which the expectoration presents, will serve to indicate, on the one hand, merely congestion of the bronchial membrane, or, on the other hand, a superinduced pulmonary affection. The existence and nature of the latter, however, are to be ascertained, not by the cough and expectoration alone, but by means of other associated symptoms and by physical signs. The accumulation of liquid within the bronchial tubes often increases the dyspnœa, and, at an advanced period, may prove the immediate cause of death by apnœa.

Hæmoptysis is a symptom which occurs in a pretty large proportion of cases of valvular lesions attended with a marked degree of engorgement of the lungs. It is due to distension of the vessels of the bronchial membrane; diminished cohesion from impaired nutrition eventuating in rupture at certain points. The amount of hemorrhage varies in different cases. It is rarely large, and often quite small. In some cases, frequent repetitions of the hæmoptysis take place. It is rare that the loss of blood is sufficient to constitute grounds for apprehension, although this symptom generally occasions alarm in the minds of patients and friends. The immediate effect is perhaps oftener salutary than otherwise, the pulmonary congestion being temporarily relieved by the direct depletion from the engorged vessels. Of the different lesions, mitral contraction is by far the most likely to give rise to bronchial hemorrhage. It occurs, however, by no means exclusively in connection with this form, but is observed in cases of mitral regurgitation, and sometimes in connection with lesions at the aortic orifice. It was formerly attributed incorrectly to hypertrophy of the right ventricle, the augmented power of the contraction of this ventricle being supposed to impel the current of blood into the pulmonary vessels with a force sufficient to produce rupture. It is not probable that this alone is ever adequate to give rise to hæmoptysis, but it is reasonable to suppose that it may exert some agency in conjunction with the valvular lesions which occasion obstruction at the left side of the heart. Dilatation, however, more than hypertrophy of the right ventricle, co-operates with the valvular lesions in the production of this symptom by contributing to retard the circulation through the lungs.

Hemorrhagic extravasation, pneumorrhagia or pulmonary apoplexy, involves the same pathological explanation as hæmoptysis, but occurs much more infrequently than the latter. It is an occasional effect of engorgement. In most of the cases in which it occurs, there exists mitral contraction. This pulmonary affection, indeed, is very rarely observed except in connection with valvular lesions which give rise to obstruction at the left side of the heart. As regards the agency of hypertrophy of the right ventricle in its production, the remarks made with reference to hæmoptysis are alike applicable. Hæmoptysis and hemorrhagic extravasation occasionally coexist, but the latter, as well as the former, occurs without the other. In proportion to the extent of solidification of lung by the extravasated blood, will the respiratory function be compromised, and dyspnœa increased. The symptoms and signs pertaining to this affection will, of course, be superadded to those which belong to pulmonary congestion. For the diagnostic points, which are not highly distinctive, the reader is referred to works treating of diseases of the respiratory system.

Pulmonary œdema is another pathological effect attributable directly to over-distension of the vessels of the lungs. This event takes place much more frequently than extravasation of blood. The liability to its occurrence, other things being equal, is proportionate to the amount of obstruction at the left side of the heart; but it is more likely to occur when weakness of the right ventricle from dilatation or fatty degeneration is superadded. A condition of the blood disposing to transudation favors its occurrence. When such a condition exists, œdema of the lungs occurs in connection with effusion in other situations, or general dropsy. Occurring alone, or irrespective of dropsical effusion elsewhere, it belongs among the events incident to an advanced stage of valvular lesions. It adds to the dyspnœa in proportion to the amount of pulmonary parenchyma involved, and also increases the cough and expectoration. In a certain proportion of cases it proves the immediate cause of death by apnœa. Its occurrence is denoted by physical signs (dulness on percussion and the subcrepitant or a fine mucous râle), which render the diagnosis easy.

The symptoms and pathological effects which have been noticed, it will be borne in mind, are produced by valvular lesions through the intervention of the pulmonary engorgement incident to these lesions. They are immediate effects of this engorgement. Valvular lesions, therefore, may exist without giving rise to these effects, so long as the lesions do not interfere materially with the pulmonary circulation. It does not follow from the fact that there is abundant evidence of the existence of valvular lesions, that these symptoms and effects will speedily occur, for lesions may exist for an indefinite period without occasioning a marked degree of congestion of the lungs. Moreover, the pulmonary circulation bears with impunity a certain amount of obstruction. As a rule, whenever events of importance referable to the respiratory system become developed, valvular lesions have existed for a considerable length of time, and have led to more or less enlargement of the heart. The heart, when enlarged, with or without lesions of the valves, encroaches on space which otherwise would have been occupied by the lungs. In this way the respiratory function is to some extent compromised. I have met with instances of collapse of the lower lobe of the left lung, apparently due to the pressure of an enlarged heart.

Certain pulmonary affections not due directly or exclusively to the congestion proceeding from valvular lesions, are more apt to occur under these circumstances, than if the latter did not exist. The lesions thus indirectly predispose to the development of these affections. Emphysema of the lungs is one of these affections. This is certainly observed among a given number of persons affected with valvular lesions, in a larger ratio than among the same number of persons free from these lesions. Without entering into a discussion of the mechanism by which pulmonary emphysema is produced, which would be here out of place, I shall simply remark that the histories of cases in which this affection is developed during the progress of valvular lesions, show that it is preceded and accompanied by bronchitis, to which it probably sustains the same relation as when it is developed irrespective of cardiac disease. Occurring as a complication of valvular lesions, it adds notably to the dyspnœa, overshadowing in some instances the cardiac symptoms. Moreover, increasing the obstruction to the pulmonary circulation, it co-operates with the impediment due to the valvular lesions in leading to enlargement of the right ventricle. It is not easy in individual cases always to determine the amount of dyspnœa and other symptoms attributable, on the one hand, to the emphysema, on the other hand, to the congestion incident to valvular lesions. This problem can only be solved approximately by endeavoring to estimate the extent to which the lungs are emphysematous, by means of diagnostic signs which it belongs to works treating of diseases of the respiratory system to consider, and also ascertaining the amount of cardiac enlargement which exists.

The congested state of the bronchial mucous membrane renders it prone to inflammation. Bronchitis is a frequent complication of valvular lesions after they have induced pulmonary engorgement. This complication occurring in persons free from antecedent disease of the lungs or heart, and limited, as is usually the case, to the larger tubes, very rarely gives rise to dyspnœa. But developed when the lungs are already congested in connection with cardiac lesions, dyspnœa becomes more or less prominent. The existence of bronchitis, disconnected from other pulmonary affections, is determined by its positive symptoms and signs, and by the absence of the diagnostic phenomena which characterize other affections. The coexistence of bronchitis not only adds to the distress incident to valvular lesions which interfere with the pulmonary circulation, but, if severe or extensive, often places the patient in immediate danger, the accumulation of mucus within the bronchial tubes, together with the diminished calibre of the tubes from swelling of the membrane, inducing suffocation. In some persons the bronchial inflammation leads to spasm of the muscular fibres of the membrane, giving rise to attacks of true asthma. As already stated, it is probably by the intervention of bronchitis that valvular lesions predispose to emphysema.

Persons affected with valvular lesions which have led to pulmonary congestion, are more prone than others to pneumonitis. The affection occurring in this connection is attended with much greater embarrassment of respiration, and disturbance of the circulation, than when it occurs as a primary disease; and it is more likely to prove fatal. Death sometimes occurs during the first stage of the disease, which is very rarely the case when pneumonitis is primary. The characteristic symptoms and signs of the affection render the fact of its coexistence, in individual cases, easily determinable.

Dropsical effusion into the pleural sacs rarely occurs to much extent independently of general dropsy. When it does take place, the compression of the lungs by the effused liquid abridges their functional capacity, aggravates the dyspnœa, and hastens a fatal issue. The effusion, when purely dropsical, exists in both sides of the chest, the quantity in one side, however, often exceeding considerably that in the other side. But, in a certain proportion of cases, pleuritic inflammation becomes developed in one side, giving rise to an abundant effusion of liquid, nearly or quite filling the affected side. In several instances which have fallen under my observation, pleurisy has occurred when the cardiac affection had not previously occasioned sufficient inconvenience to prevent the patients from continuing laborious occupations. The inflammation is subacute. The affection is developed, as cases of chronic pleurisy frequently are, with little or no pain, the chief subject of complaint being dyspnœa. So slight are the symptoms referable to the lungs, exclusive of dyspnœa, that if the attention of the physician be directed to the heart, there is a liability of overlooking the pleuritic effusion, unless pains are taken to explore the chest for the signs of the latter. On examination after death the liquid effused is not clear, as in cases of hydrothorax, but it is not notably turbid, and the exudation of lymph is not abundant. The inflammatory action has a low grade of intensity. This is true of the instances that have come under my observation. The effused liquid is less likely to be removed by absorption than in cases in which chronic pleurisy is not connected with valvular lesions. A fatal termination is hastened by this complication, and in some instances death takes place very soon after its occurrence. The physical signs of an abundant pleuritic effusion are so simple and well marked, that if overlooked by those who avail themselves of physical exploration of the chest, it must be from inattention. Chronic pleurisy is developed in some cases in which valvular lesions are associated with albuminuria and Bright's diseases, but it occurs when the kidneys are free from disease.

SYMPTOMS AND PATHOLOGICAL EFFECTS REFERABLE TO THE NERVOUS SYSTEM.

The majority of cases of valvular lesions end without having given rise to prominent symptoms or important pathological effects referable to the nervous system. This statement is at variance with the notions generally entertained and inculcated by some writers. It is a common impression that various symptoms denoting cerebral disorder, such as cephalalgia, vertigo, tinnitus aurium, muscæ volitantes, &c., &c., are usually observed, sooner or later, during the progress of cardiac disease. These symptoms are often observed in persons not affected with disease of the heart, and, hence, would possess small diagnostic significance were they more frequently present; but, the truth is, they occur in only a small proportion of cases, at least in a marked degree. Apoplexy and paralysis are events less common than has been heretofore supposed. It is then hardly necessary to consider at much length the mooted question, whether cerebral phenomena and complications proceed from the abnormal power of an hypertrophied left ventricle, or from the obstruction occasioned by over-distension of the right auricle. It is conceivable that either and both may conduce, in certain instances, to congestion, extravasation of blood, and serous transudation; but clinical facts show that, separately or combined, they very rarely produce disorder of the cerebral circulation sufficient to occasion great inconvenience to the patient or lead to serious results. Of seventy-two fatal cases of lesions affecting the mitral and aortic orifice, separately or together, which I have analyzed with reference to this point, the histories of fifty-five present no symptoms or events of importance pertaining to the brain.

Valvular lesions, accompanied by enlargement of the heart, have been supposed to involve a strong liability to apoplexy. This opinion was held by Hope. Apoplexy occurs in a small proportion of cases of valvular disease. Of 117 cases analyzed with reference to this event it took place in ten. But even in the few cases in which apoplexy and valvular lesions are associated, circumstances go to show that there often exists only a remote and contingent pathological connection between them. Of the ten cases just mentioned, in eight the age of the patients was forty or more years. In one case it was eighty, in one, sixty-six, in one, fifty-five, in one, fifty-two, in one, over sixty, in one, forty, and in one case the age was not noted. The ages thus were within the period when apoplexy, irrespective of disease of the heart, is most apt to occur. In some of the cases, the cerebral arteries were found to have become calcified. In one of the cases, the patient being forty-one years of age, the valvular affection was trivial, accompanied by slight enlargement. It is not infrequently the case when, from the coincidence of apoplexy and some morbid appearances of the valves, the former is supposed to be dependent on the latter, that the valvular lesions are not sufficient to have occasioned much disturbance of the circulation. On the other hand, how rarely does apoplexy occur when there exist over-distension and dilatation of the right auricle, and in cases of considerable hypertrophy of the left ventricle! In view of these facts, it is reasonable to conclude that apoplexy is very rarely due, directly or exclusively, to the condition of the heart, but that the changes which the cerebral vessels undergo, or other circumstances, generally play an important part in its production. This is not to deny more or less agency to the heart in certain cases. And of the two conditions which tend directly to affect the circulation in the brain, namely, obstruction at the right side of the heart, and hypertrophy of the left ventricle, the former must be considered as most likely to lead to serious results. Hypertrophy of the left ventricle is generally associated with either obstructive or regurgitant lesions at the aortic or mitral orifice. An effect of these lesions is either to diminish the quantity of blood sent to the brain, or to break the force of the ventricle upon the arterial current. The latter obtains when the aortic orifice is contracted, and the former in cases of aortic regurgitation and of mitral lesions, whether obstructive or regurgitant. The opinion held by Hope and others that apoplexy sustains a direct pathological connection with hypertrophy of the left ventricle, is disproved on rational grounds as well as by clinical evidence.

Apoplexy occurring in connection with cardiac lesions generally depends on extravasation of blood. Under these circumstances, paralysis, of course, ensues; if the apoplectic attack do not prove suddenly fatal, the patient is found to be hemiplegic. Paralysis sometimes occurs without having been preceded by apoplexy. Both apoplexy and hemiplegia may proceed from a cause emanating from the heart, independently of either an impediment at the right auricle or hypertrophy of the left ventricle. Reference is now made to an event already noticed under the head of pathological effects referable to the circulation, namely, embolism. Vegetations or masses of fibrin from within the cavities of the left side of the heart being detached are liable to become fixed in an artery of the brain, and give rise to apoplectic seizures with hemiplegia, or to the latter without the former. In this way, valvular lesions sustain toward these cerebral affections a direct causative relation. This explanation of apoplexy and paralysis associated with valvular lesions is rendered probable in cases in which, from the age of the patient, fatty or calcareous degeneration of the cerebral arteries is not likely to have occurred, and when there does not exist a notable degree of obstruction at the right side of the heart. After death, embolism is to be suspected when the aortic or mitral valves are found to present vegetations or excrescences, some of which are loosely attached. But the proof consists in finding impacted in an arterial trunk of the brain an embolus which is found, on microscopical examination, to be identical with vegetations or masses of fibrin on the valves in the left side of the heart. Complete recovery from paralysis is a ground for suspecting that it originated in arterial obstruction rather than in extravasation, the restoration of power over the paralyzed muscles, when the latter occurs, being never complete.

Arterial obstruction gives rise to apoplectic phenomena and paralysis by lessening the supply of blood to certain portions of the cerebral substance. The pathological condition induced, therefore, is the opposite of that incident to an impediment to the circulation at the right side of the heart. In the one case a part of the brain suffers from anæmia; in the other case the whole brain is congested. Softening of the cerebral substance attributable to impaired nutrition from the defective supply of blood may follow the interruption of the circulation by fibrinous plugs or emboli. Law, of Dublin, attached considerable importance to the defective supply of blood to the whole brain as a result of either mitral or aortic obstruction. In these lesions more especially, but to a considerable extent also in those attended by regurgitation, the stream of blood propelled into the aorta and cerebral arteries is obviously lessened. The brain receives with each contraction of the left ventricle a quantity of blood less than when the valves and orifices are free from disease. Law attributed cerebral softening in some instances to this cause.[1] The importance attached by this writer, however, to an anæmic state of the brain, as thus induced, is not sustained by clinical observation, since it is only in a small proportion of the cases attended with more or less obstruction or regurgitation, or both, at either the aortic or mitral orifice, that cerebral symptoms denoting any important pathological condition are manifested. Cases of extreme aortic and mitral contraction proceed to a fatal termination, the histories not containing aught which indicates that the brain has suffered from a deficient supply of blood.

Attacks of pseudo-apoplexy, that is, of coma, more or less complete, continuing for a certain period and passing off without paralysis, have been observed in cases of valvular lesions. They have been already described in treating of fatty degeneration of the heart. They may depend on the latter change coexisting with valvular lesions; but, as remarked in connection with the subject of fatty degeneration, the pathological relation between these attacks and the existence of any organic disease of the heart cannot be considered as established.

Aside from apoplexy and paralysis, various symptoms already mentioned, namely, pain, vertigo, tinnitus, &c., are occasionally associated with valvular lesions. Apoplexy and paralysis depending either on an extravasation which involves a morbid condition of the cerebral vessels, or on arterial obstruction from emboli, are usually not preceded by premonitions referable to the brain. Clinical observation shows that a liability to these affections is not to be predicated on the symptoms just referred to. This is a practical point to be borne in mind in order that needless apprehensions be not entertained on the part of physician or patient, and measures employed, with a view of warding off an attack of apoplexy or paralysis, which, being uncalled for, will be likely to be not only unnecessary, but injudicious. In the cases in which there is of necessity more or less cerebral congestion, the superficial veins of the neck being swelled or pulsating, marked cerebral symptoms are by no means uniformly present. Headache, dulness of the intellect, listlessness, drowsiness, &c., are symptoms which, in a certain proportion of cases of this description, are more or less marked, and are probably due to abnormal fulness of the cerebral veins. These symptoms of cerebral oppression are sometimes marked in cases in which, either from obstruction at the right side of the heart or imperfect oxygenation of the blood, the prolabia and surface of the body present a livid appearance.

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[1] Dublin Journal of Medicine, May, 1840.

The sleep of patients affected with cardiac disease is frequently imperfect. They complain often of frightful dreams. This is generally associated with dyspnœa, and appears to be owing to disturbed respiration rather than to disordered cerebral circulation. Moaning in sleep is a symptom observed in some cases when the patient is not wakeful nor conscious of any morbid sensations.

A symptom which may be included among the events referable either to the nervous or respiratory system, is noticed in some cases, namely, a choking sensation analogous to that experienced in painful emotions when an effort is made to refrain from weeping. This is not of frequent occurrence, but it has been prominent in several instances among the cases that have come under my observation. It is associated with more or less dyspnœa.

The mental condition of patients affected with organic disease of heart, may be noticed in this connection. The contrast presented in this respect, with patients affected with merely functional disorder, has been already referred to. Persons with organic disease which has given rise to grave symptoms, such as palpitation, dyspnœa, dropsy, &c., are generally free from excitement and apprehension. They often seem to be remarkably indifferent or apathetic. They are not agitated when made acquainted with the fact that they have organic disease of the heart. They are sometimes incredulous as to the seat of the disease, and are disposed to attribute their ailments to the liver, lungs, or stomach. The mental condition, in short, is quite the reverse of that usually associated with affections purely functional. A comparison of the characters pertaining to the feelings, which belong to the history of organic lesions of the heart, with those observed in some other diseases, affords a striking illustration of the great difference in the effects produced on the mind by different morbid conditions irrespective of cerebral disease.

Some degree of mental aberration is occasionally observed toward the close of life in cases of valvular lesions, but delirium cannot be reckoned among the events belonging to their natural history.

SYMPTOMS AND PATHOLOGICAL EFFECTS REFERABLE TO THE DIGESTIVE SYSTEM AND NUTRITION.

The phenomena manifested in connection with the digestive apparatus, in cases of valvular lesions, proceed from congestion of the systemic venous system. Assuming the lesions to be either mitral or aortic, or both, congestion of this order of vessels depends on the effects of these lesions on the right side of the heart. It may be stated that, as a rule, the systemic congestion is not sufficient to give rise to important symptoms or pathological effects until dilatation of the right ventricle has taken place, involving over-distension of the right auricle, and, in certain cases, tricuspid regurgitation. The impediment to the free admission of blood from the venæ cavæ into the right auricle, occasions cerebral congestion, as has just been seen. The congestion throughout the body thus induced, as has also been seen, gives rise to venous turgescence and general dropsy. The abdominal viscera indirectly participate in the effects of this impediment at the right ventricle, owing to their vascular relations to the venæ cavæ being through the intervention of the portal system. In view of the anatomical peculiarities of the latter, it is obvious that, of the organs comprising the abdominal viscera, the liver is first affected by an obstruction at the right side of the heart. The radicles of the hepatic veins (the intralobular veins) are the first of the different orders of vessels contained in this viscus, to show engorgement. The terminal branches of the portal vein (the interlobular veins) are next affected. The appearances after death indicate whether either or bóth of these sets of vessels are unduly congested. The pressure of the portal branches, or interlobular veins, on the biliary tubes may occasion an undue accumulation of bile in the latter. Sections of the organ then present that peculiar aspect commonly known as the "nutmeg liver." Extending beyond the liver to the portal vein and its radicles, the congestion affects finally the stomach and intestines, the spleen and the pancreas. Congestion of these organs is a secondary effect due directly to the mechanical obstacle to the passage of blood through the liver. The successive steps, thus, in the series of congestive effects dependent on valvular lesions are: Obstruction or regurgitation at either the mitral or aortic orifice, or at both situations; dilatation of the right ventricle following engorgement of the pulmonary vessels; over-distension of the right auricle, with or without tricuspid regurgitation, involving an impediment to the free transmission of blood from the venæ cavæ; congestion of the hepatic vein, and its radicles, the intralobular veins; congestion of the terminal branches of the portal vein, or the interlobular veins; congestion of the vena portæ and its radicles in the abdominal viscera, these vessels furnishing the blood for the portal circulation.

Clinical observation shows, as might rationally be anticipated, that the phenomena due to engorgement of the abdominal viscera are developed, in the order of time, consecutively to the general effects of congestion of the systemic veins. It is rarely the case that the former occur to much extent until the obstruction at the right side of the heart is sufficient to give rise to more or less general dropsy. As a general remark, symptoms and pathological effects referable to the digestive system do not hold a prominent place among the events which belong to the natural history of valvular lesions. This statement is made after analyzing the histories of one hundred cases, extending in seventy to the period of death. In a large majority of these histories, nothing of importance was noted with reference to the digestive system.

Enlargement of the liver is an occasional effect incident to valvular lesions, as well as to enlargement of the heart without these lesions. This has been already noticed in connection with enlargement of the heart. It is more correct to say that this is an effect of enlargement affecting the right side of the heart, either with or without the coexistence of valvular lesions, the latter inducing the effect through the intervention of the cardiac enlargement, as has just been stated. The augmented size of the liver is in some instances remarkable, and its variations in size at different periods is not less striking. The enlargement is due simply to the excessive accumulation of blood in the vessels of the organ. Jaundice is an occasional symptom. It is met with, however, in a small proportion of cases.

Cirrhosis, contrary to an impression heretofore common, is not a frequent complication of valvular affections of the heart. The congested state of the liver incident to these affections does not seem to tend to its production. The concurrence of these affections with this structural change of the liver is so infrequent as hardly to afford ground for the opinion that there exists between the two any pathological connection. When associated, it is probably simply a coincidence. Becquerel reported the existence of cardiac disease in twenty-one of forty-two cases of cirrhosis which he analyzed, the former being deemed to have occurred prior to the latter.[1] But in more than one-half of the twenty-one cases, he regarded the cirrhosis as in the first degree, giving rise to no symptoms of importance. These statistics, as remarked by Budd, are to be accounted for on the supposition that the abnormal appearances due to congestion of the different sets of vessels were confounded with the commencement of cirrhosis. French, on the other hand, found disease of the heart in only four of thirty-six cases of cirrhosis. In only five of thirty-two cases which I have analyzed was there evidence of valvular lesions with enlargement of the heart. The event denoting coexisting cirrhosis, is hydroperitoneum in a degree disproportionate to the general dropsy.

The various phenomena included in the term indigestion, which are common to a great number of affections, may be absent or present, and more or less prominent in cases of valvular lesions. But, in a large proportion of cases they do not occur in a marked degree, at least during the greater portion of the time occupied by the progress of the lesions before a fatal issue takes place. Patients who suffer from the distressing effects of obstructive or regurgitant lesions, together with enlargement of the heart, often preserve their appetite, and the ingestion of food occasions no inconvenience. This, in fact, is usually the case, so that disordered digestion cannot be considered to characterize organic affections of the heart. The disorders which are observed in an advanced stage, after general dropsy has taken place, are probably due, in part at least, to congestion of the gastric mucous membrane. Gastrorrhagia is one of the rare effects, occurring sometimes when cirrhosis of the liver does not coexist.

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[1] Archives Générales de Médecine, 1840. Budd on Diseases of the Liver, second Am. ed., p. 148.

Intestinal flux, or enterorrhœa, is another infrequent symptom, the serous transudation taking a direction through the mucous tissue, instead of, or in addition to, the more common direction into the peritoneal cavity.

Hemorrhage from the bowels is to be ranked in the same category. The same is to be said of hemorrhoids. In this connection may be mentioned epistaxis, which occurs more frequently as a result of obstruction at the right side of the heart than hemorrhage in any other situation. It has been supposed that the escape of blood from the nostrils may, in some instances, prevent extravasation into the brain, or other serious effects of cerebral congestion, by relieving the vessels, in a measure, of their over-accumulation.

Enlargement of the spleen, due exclusively to the congestion of the portal system dependent on cardiac obstruction, is rare. It is, however, to be reckoned among the effects which are occasionally observed.

The functions of nutrition, applying this term to the processes of the growth and repair of the tissues, are much less affected than, on rational grounds, would be anticipated, even when the lesions of the valves have led to enlargement of the heart and much disturbance of the circulation. Patients suffering from the distressing effects of cardiac disease, namely, dyspnœa, palpitation, œdema, &c., often do not emaciate. When these effects occur in early life, the development of the body is sometimes not remarkably impaired. Even at an advanced stage, considerable embonpoint is frequently maintained. It is not uncommon to find the evidences of lesions, which must have existed for a long time, in persons whose general aspect denotes excellent health. So far from diminished nutrition being one of the pathological effects of valvular lesions, they are rather to be characterized by the absence of notable deterioration in this respect. In cases in which the origin of valvular lesions dates in early life, and enlargement of the heart takes place before puberty, the body may attain to a full development.

SYMPTOMS AND PATHOLOGICAL EFFECTS REFERABLE TO THE GENITO-URINARY SYSTEM.

The renal or emulgent veins terminating in the vena cava descendens, the kidneys must participate in the congestion of the systemic venous system arising from an impediment at the right side of the heart. These organs are affected more directly than the abdominal viscera which are tributary to the portal vein. So soon as valvular lesions have led to the anatomical conditions involving an obstruction extending to the venæ cavæ and their branches, renal engorgement necessarily ensues. Congestion of these organs is generally observed in examinations after death in cases of valvular disease accompanied by dilatation of the cavities of the right side of the heart. Venous congestion, under these circumstances, does not uniformly occasion a greater flow of urine than in health. Indeed, the quantity of urine is oftener diminished than increased, a fact going to show that the diuresis depends on the amount of blood conveyed to the kidneys by the arteries, or on conditions pertaining to the blood itself, rather than on accumulation in the renal veins. The urine is frequently scanty, even when the venous obstruction is sufficient to give rise to general dropsy. The solid constituents are relatively augmented; in other words, the density of the urine is greater than in health. The lithatic deposits are often abundant. The presence of albumen is not infrequently shown by the appropriate tests. If the kidneys have not undergone structural change, the quantity of albumen is usually slight. It may be found, on repeated examinations in the same case, sometimes present, and at other times absent. The quantity at different times may be found to fluctuate. Albuminuria in these cases may be fairly attributed to the mechanical pressure incident to venous congestion. It does not constitute evidence of structural changes of the kidney or Bright's diseases when it is in small quantity, transient in duration, and notably fluctuating. Moreover, hyaline casts are wanting when albuminuria is only a symptom of disease of the heart, and the specific gravity of the urine, instead of being diminished, is usually increased.

The degenerations of structure included under the name Bright's diseases, are sometimes associated with valvular lesions of the heart. The frequency of this combination, however, is less than is generally supposed. Accepting, on the one hand, as the evidence of coexisting Bright's diseases, either an abundant quantity of albumen constantly present in the urine during life, or well-marked anatomical characters observed after death, and, on the other hand, including only those cases of cardiac disease which involve obstruction or regurgitation, or both, the two affections are not very often united. It may perhaps be doubted whether they are associated sufficiently often to establish any direct pathological connection between them. Assuming the existence of such a connection, however, some have attributed the renal affection to the cardiac disease, and others have thought that the valvular lesions were due to the condition of the kidneys. They who accept the first of these suppositions refer the development of structural change in the kidneys to the congested state of these organs. But it is by no means certain that mere congestion is adequate to produce this result. The second supposition is more tenable. Pericarditis and other serous inflammations are not infrequently developed in the course of Bright's diseases; and it may be reasonably argued that endocarditis is occasionally incidental to the latter. Clinical observation has not, as yet, fully confirmed the correctness of this analogical argument.

The coexistence of structural degeneration of the kidney is shown, as already intimated, by the degree and constancy of the albuminuria, and by the different varieties of casts of the uriniferous tubes, which the sediment of the urine is found to contain when subjected to microscopical examination. The tendency to general dropsy is augmented by this complication; renal and cardiac dropsy are, in fact, combined. It is needless to say that the danger is vastly increased by the addition of a renal disease which renders the patient liable to other accidents than those incident to the cardiac disease, and aggravates some of the most important of the pathological effects of the latter.

As regards the generative functions, the histories of valvular affections which I have collected furnish no facts of significance or importance. I have observed, in cases in which lesions had existed for a considerable period before puberty, that the genital organs, including, in females, the mammary gland, have attained to a full development.

SYMPTOMS AND PATHOLOGICAL EFFECTS REFERABLE TO THE COUNTENANCE AND EXTERNAL APPEARANCE OF THE BODY.

The characters pertaining to the countenance have, for the most part, been already incidentally mentioned. Lividity of the prolabia and face, which may be apparent over the whole surface of the body, denotes either venous congestion or imperfect oxygenation of the blood. The latter is incident to the pathological effects taking place in the lungs; the former, to obstruction at the right side of the heart. But both conditions may be conjoined. Cyanosis dependent on congenital malformations will be considered hereafter. A dusky hue of the face, combined with œdema, is quite distinctive of cardiac, as contrasted with renal, dropsy. The experienced clinical observer is able to make this differential diagnosis with much precision at a glance. When the lividity is marked, and the œdema considerable, the face presents an appearance like that of a cadaver after strangulation. The expression is sometimes so much altered that the patient is scarcely recognized by familiar friends. Urgent dyspnœa induces an expression of great anxiety and distress. The painful spectacle presented by a case of extreme suffering from so-called cardiac asthma is thus vividly portrayed by Hope: "Incapable of lying down, he is seen for weeks, and even for months together, either reclining in the semi-erect posture supported by pillows, or sitting with the trunk bent forward, and the elbows or forearms resting on the drawn-up knees. The latter position he assumes when attacked by a paroxysm of dyspnœa; sometimes, however, extending the arms against the bed on either side, to afford a firmer fulcrum for the muscles of respiration. With eyes wildly expanding and starting, eyebrows raised, nostrils dilated, a ghastly and haggard countenance, and the head thrown back at every inspiration, he casts around a hurried, distracted look of horror, of anguish, and of supplication: now imploring in plaintive moans, or quick, broken accents and half-stifled voice, the assistance already often lavished in vain; now upbraiding the impotency of medicine, and now, in an agony of despair, drooping his head on his chest, and muttering a fervent invocation for death to put a period to his sufferings. For a few hours—perhaps only for a few moments—he tastes an interval of delicious respite, which cheers him with the hope that the worst is over, and that his recovery is at hand. Soon that hope vanishes. From a slumber fraught with the horrors of a hideous dream, he starts up with a wild exclamation that 'it is returning.' At length, after reiterated recurrences of the same attacks, the muscles of respiration, subdued by efforts which the instinct of self-preservation alone renders them capable of, participate in the general exhaustion, and refuse to perform their function. The patient gasps, sinks, and expires."[1] Happily the fearful intensity of suffering depicted in the foregoing sketch characterizes a small proportion only of the cases of valvular disease which proceed to a fatal termination.

Some cases of valvular disease are characterized by pallor of the complexion. The coexistence of Bright's diseases is likely to lead to this effect. But it is observed in some instances when the kidneys are not affected. It then depends on alterations of the blood proceeding from other causes. An anæmic condition is incident, in a certain proportion of cases, to cardiac disease uncomplicated with an affection of the kidneys. Analysis of the blood shows a notable deficiency of albumen, together with a reduction in the relative proportion of blood corpuscles and fibrin. This condition of the blood is important in connection with therapeutical measures. It will be aggravated by depletion, and to remove it by appropriate treatment, if practicable, should be an important object with the practitioner.

The accumulation of blood in the right chambers of the heart induces, in addition to abnormal fulness of the superficial veins, a congestive state of the capillary vessels, causing the surface of the body to present an appearance like that produced by exposure to cold. The redness disappears on pressure, and returns, more or less slowly, after the pressure is removed. The appearance is not unlike that observed in typhus and typhoid fever, although the *rationale* is by no means the same. Erythema affecting portions of the surface occurs in some cases not associated with œdema. The lower extremities are most apt to be affected. I have met with an instance in which the extremities of the fingers and a portion of the palms presented permanently an erythematic redness. On the other hand, in a patient with considerable mitral regurgitation, the fingers at times were bloodless, being as pallid and cold as those of a corpse.

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[1] On Diseases of the Heart, Am. ed., p. 382.

CHAPTER IV.

PHYSICAL SIGNS, DIAGNOSIS, AND TREATMENT OF VALVULAR LESIONS.

Endocardial or valvular murmurs—Distinction between endocardial and exocardial murmurs—Vascular murmurs—Distinction between inorganic and organic murmurs—Soft and rough murmurs—Musical murmurs—Enumeration of abnormal conditions giving rise to endocardial murmurs, organic and inorganic—Valvular lesions involving obstruction or regurgitation, or both, generally accompanied by a murmur—Circumstances enabling the auscultator to determine whether lesions involve obstruction, or regurgitation, or both—Mitral direct, or systolic murmur—Mitral regurgitant or diastolic murmur—Aortic direct or systolic murmur—Aortic regurgitant or diastolic murmur—Localization of systolic murmurs—Localization of diastolic murmurs—Recapitulation of points involved in the localization of systolic and diastolic murmurs—Pathological import of organic endocardial murmurs—Inorganic murmurs—Abnormal modifications of the heart-sounds in cases of valvular lesions—Purring tremor—Diagnostic characters of mitral, aortic, tricuspid, and pulmonic lesions—Sudden death in cases of valvular lesions—Treatment of valvular lesions.

THE physical signs of lesions affecting the valves and orifices of the heart are to be considered preparatory to entering on the consideration of the diagnosis of these lesions. It is chiefly by means of auscultatory signs that the existence and seat of valvular lesions are determined during life. The symptoms and pathological effects, which were considered in the last chapter, afford important aid in the diagnosis, but, alone, they often fail in furnishing positive evidence that the valves or orifices are affected, and still less do they indicate the particular situation of existing lesions. To the study of the auscultatory signs in connection with researches on the mechanism of the heart's action, practical medicine is indebted for the great perfection to which the diagnosis of cardiac affections has attained within late years. Here, as in other instances, the physical phenomena have a negative, as well as positive, application in diagnosis; that is to say, while they constitute evidence of the presence of certain lesions, their absence is, in general, proof that lesions do not exist.

In the practice of auscultation with reference to the cardiac murmurs, as well as to the heart-sounds, the stethoscope is much to be preferred to the application of the ear directly to the chest. The latter will often suffice, but the study of the murmurs, as regards their character, the situations in which their intensity is greatest, and their relations to the sounds of the heart, is much more satisfactory by means of the stethoscope. And here, too, the binaural stethoscope is superior, beyond comparison, to any of the instruments which have been heretofore in use. By means of the binaural stethoscope murmurs may be discovered which would be overlooked were immediate auscultation only to be employed.

The auscultatory signs of valvular lesions may be arranged into two classes, namely: *first*, new or adventitious sounds, which are called *murmurs;* and *second*, abnormal modifications of the sounds of the heart. The first class, or the murmurs, from their practical importance, require to be considered at considerable length.

ENDOCARDIAL OR VALVULAR MURMURS.

All adventitious sounds, dependent on the movements of the heart, and either replacing or being superadded to the normal heart-sounds, are distinguished as *cardiac murmurs*. Strictly speaking, these murmurs are heart-sounds, but, for the sake of distinction, the latter term is conventionally restricted to the normal sounds of the heart, together with their abnormal modifications. The French word *bruit* is often used by English and American writers. This term with French writers is synonymous with murmur. The latter term is sufficiently distinctive and convenient, so that it is quite needless, in this instance, to have recourse to a foreign tongue.

Cardiac murmurs originate either within the heart, or on the peripheral surface of the organ. Latham proposed to distinguish those produced within the heart as *endocardial*, and those produced upon the surface as *exocardial* murmurs. These names are frequently used, and they are sufficiently distinctive. It is, however, convenient sometimes to include among endocardial murmurs those produced in the aorta and pulmonary artery in close proximity to the heart. Murmurs produced within vessels more or less removed from the heart, may be called *vascular murmurs*. The latter may originate either within the arteries or veins. The endocardial and vascular murmurs require for their production the passage of currents of blood through the cavities of the heart, its orifices, or into the bloodvessels. These murmurs are often called *bellows murmurs* (*bruit de soufflet*) from the resemblance of the sound to that produced by the expulsion of air from the nozzle of an ordinary bellows. This resemblance is often striking, and holds good in the majority of instances; but some intra-cardiac and vascular murmurs are very inappropriately called bellows-murmurs, resembling sounds other than that implied by this name. It is, however, to be borne in mind that all adventitious sounds produced within the heart and vascular system are often conventionally known as bellows murmurs. This name was applied to them by the illustrious discoverer of auscultation, Laennec, by whom they were first described.

Exocardial murmurs are caused by the rubbing together of the visceral and the parietal surface of the pericardium, and sometimes by the heart impinging against the neighboring parts exterior to the pericardium. These murmurs are usually called, from the manner of their production, *attrition* or *friction murmurs*. They will be considered hereafter in connection with the diseases affecting the pericardium.

In treating of endocardial murmurs, the practical points to be considered relate to the different characters which they present, the morbid conditions which they denote, their significance and value as signs of disease, and their application to the diagnosis of valvular affections. These murmurs may be produced within the cavities of the heart, at the auriculo-ventricular or the ventriculo-arterial orifices, and within the aorta or pulmonary artery near the junction of these vessels with the ventricles. Strictly speaking, murmurs produced in the last-named situations are not endocardial, but it is sometimes convenient to include them in the same class. It is practicable often, if not generally, to determine from which of the cavities, orifices, or vessels mentioned, emanate the murmurs heard in individual cases. The importance of this localization, as pointing to the seat of the lesions which occasion the murmurs, is obvious. The special objects to which the study of this class of murmurs has been subservient, may be stated as follows: To determine the existence or nonexistence of valvular disease; to determine the particular situation of structural lesions; to determine the character of lesions and certain of their effects especially on the blood-currents through the different orifices.

Endocardial murmurs are not always due to lesions of structure or organic disease. They occur as a result of certain blood-changes and of functional disorder of the heart. The latter are distinguished as *inorganic murmurs*, while those dependent on structural changes are called *organic murmurs*. It is of great importance to discriminate, in practice, between organic and inorganic murmurs. With proper knowledge and care this can generally be done. The points involved in the discrimination are to be considered. I shall, however, first consider the organic murmurs, and afterward point out the means of distinguishing from them the murmurs which are inorganic. It is then to be understood that, for the present, reference is had exclusively to organic murmurs.

Organic murmurs, as regards their sensible characters, differ in a marked degree in different cases. In the majority of cases they resemble, as already stated, a bellows-sound. Murmurs of this kind are said to be *soft murmurs*. They vary greatly in intensity, pitch, and duration. In some instances they are so feeble as to be just appreciable; in other instances they are so loud as to be heard over the whole chest, and they are sometimes perceived by the patient, especially in the night-time. In several cases that have come under my observation, patients have accurately described the sounds which had attracted their attention before any exploration of the chest had been made. They are sometimes heard by others at a distance from the chest. I have known instances where this has occurred to persons occupying the same bed with patients affected with disease of the heart. It may not be amiss to mention, in this connection, that physicians and medical students not infrequently imagine they discover, during the night, a cardiac bellows murmur in their own persons, mistaking for it a sound produced by the movement of the head on the pillow synchronously with the ventricular systole, or, possibly, by the current of blood in the cerebral arteries. Between the extremes of feebleness and loudness, different cases present every degree of gradation as respects intensity. The pitch varies within certain limits. Bouillaud first proposed the plan of representing the pitch of bellows murmurs by whispered words and letters, which is more convenient than verbal descriptions. The highest pitch, unless the sound become musical, may be represented by the letter S, and a lower grade, but still acute, by the letter R. When the pitch is low it is often represented by the word WHO, and when still more grave by the word AWE. These letters and words were selected for this purpose by Hope. The pitch is sometimes a point of importance, but it has far less significance than was supposed by the author just named. The duration of bellows murmurs is by no means uniform in different cases. They are sometimes extremely brief, resembling the shortest possible puff, and in other cases more or less prolonged.

Murmurs which lack the softness of those just referred to, and which bear but a remote resemblance, or none whatever, to a bellows sound, are distinguished as *harsh* or *rough murmurs*. These also differ greatly in their sensible characters. Different varieties have been described and named from their resemblance to certain sounds. Thus, French writers recognize *filing*, *grating*, and *rasping* murmurs (*bruit de lime*, *bruit de râpe*, *bruit de scie*, &c.), comparing the sounds to those produced by filing and rasping wood. In a case under my observation, the sound was precisely like the croaking of a frog. In another case, the murmur resembled so much a groaning sound that I supposed at first it came from the mouth of the patient. The rough murmurs, in different cases, suggest various comparisons. The varieties, however, are of very little practical consequence. They have not, severally, any special significance. It suffices to consider them simply as presenting different modifications and degrees of roughness, the latter being the only distinctive feature worthy of being noted. The distinction, indeed, between roughness and softness is not of great importance in a practical point of view, beyond the fact that the former denotes, as a rule, structural lesions. The absence of roughness, however, is by no means evidence that structural lesions do not exist. It is stated by some writers that a rough murmur indicates something more than the existence of structural lesions, namely, the presence of calcareous deposit on the valves, orifices, or lining membrane of the heart-cavities or vessels. This statement is not correct. I have met with cases in which the murmur was notably rough and no calcareous deposit was found after death. On the other hand, in cases in which the deposit is abundant, the murmur is frequently devoid of roughness. Rough murmurs, as a rule, are more intense than soft murmurs. They are oftener perceived by the patient. In a case in which the murmur resembled the croaking of a frog, the sound was accurately described by the patient, and was distinctly heard when the ear was in close proximity to the chest, but not in actual contact. The duration of rough, as of soft, murmurs varies considerably in different cases, but they are rarely, if ever, so brief as the latter in the instances in which a short puff only is heard. A soft murmur in some cases during the progress of disease is converted into a rough murmur; and the converse of this also occurs. A murmur may be soft when the action of the heart is feeble or moderately strong, and become rough when the organ is excited into greater activity; and, conversely, it is possible that a murmur which is soft when the heart acts with violence, may become rough when the organ is more tranquil.

Murmurs sometimes have a musical intonation. The sounds are compared to the sibilant râle, the cooing of a dove, the whining of a puppy, &c. (*bruit de sifflement*, *bruit sibilant*, *bruit de roucoulement*, *de piaulement*, &c.) These are as much less frequent than the rough murmurs as the latter are more infrequent than soft murmurs. They are interesting chiefly as clinical curiosities. They have no special pathological or diagnostic significance, except that they denote the existence of organic disease. They are preceded by, and may give place to, the bellows murmur. They may alternate with the latter in different conditions of the heart's action. As remarked by Bouillaud and Hope, it is as intelligible that a bellows murmur may be transformed into a musical tone as that a change in the disposition of the lips changes a blowing into a whistling sound.

Endocardial murmurs are sometimes notably affected by the respiratory movements. They are sometimes much more intense at the end of an inspiratory act than at the end of expiration. They may be appreciable only at the end of inspiration. On the other hand, they are sometimes louder, and they may be appreciable only at the end of, or during, the act of expiration. Murmurs produced at the mitral orifice are especially those which are thus liable to be affected by respiration.

This will suffice for a general description of endocardial or valvular organic murmurs. Their relations to the two sounds of the heart and to the different currents of blood, the different situations to which they may be limited, or in which they are heard with their maximum of intensity, and the directions in which they are transmitted, are of far greater importance, as regards diagnosis, than their intrinsic characters. To these practical points attention will now be directed.

The passage of the blood through the cavities and orifices of the heart and the large vessels, in health, takes place noiselessly; nothing is heard, except the normal heart-sounds which have been considered.[1] The bulk of the heart, the capacity of its cavities, the smoothness of the endocardium, the size of the orifices and vessels, the protection afforded by the valves against regurgitating currents, and the quality of the blood, are all so nicely harmonized that the circulation is unattended by a murmur unless abnormal conditions of some kind exist. The morbid changes which may give rise to adventitious sounds are various. The presence of a murmur involves only the fact that there is something abnormal. It does not indicate the seat or nature of the change that has taken place, until certain contingent circumstances are taken into account. Of the abnormal conditions which clinical observation has shown to be productive of murmurs, the more important are the following: roughness of the endocardium and of the membrane lining the aorta or pulmonary artery; the presence of morbid growths, products, or deposits, which interrupt or disturb the current of blood, and the projection into the current of rigid, unyielding valves; contraction of the auriculo-ventricular or the ventriculo-arterial orifices; dilatation and contraction of the large vessels connected with the heart; insufficiency of the valves designed to protect the orifices just named, due either to lesions affecting the valves or to dilatation of the orifices; aneurismal dilatation of a portion of one of the ventricles; dilatation of the whole ventricle, rendering the size of the cavity disproportionate to the quantity of blood; the formation of clots in the ventricles; certain alterations in the composition of the blood; sudden diminution in the circulating mass of blood; functional disorder of the action of the heart; communication between the two ventricles, and other congenital malformations. In this list of abnormal conditions, the alteration in the composition of the blood, sudden diminution in the circulating mass, and functional disorder of the heart, are to be distinguished as *inorganic*, that is, structural lesions of the heart are not involved. Murmurs due to these conditions are therefore *inorganic murmurs.*

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[1] Chap. I, page 58 *et seq.*

Murmurs dependent on any of the other conditions enumerated are *organic murmurs.*

The physical conditions on which, in the great majority of cases, organic murmurs are dependent are classed under the head of *valvular lesions* which were described in the preceding chapter. Under this head are embraced the greater part of the conditions just enumerated. The morbid appearances which the valves and orifices present in different cases, as has been seen, are extremely diversified, consisting of various forms and degrees of roughness from calcareous deposits; vegetations of greater or less size, and more or less numerous, and variously disposed; thickening on the one hand, and attenuation, rupture, perforations, and a cribriform condition on the other hand; crumpling and contraction to a greater or less extent; cartilaginous stiffness and ossific rigidity; disruption of tendinous cords; adhesion of the valves to each other and to the inner surface of the heart or to the vessels; enlargement of the orifices, congenital deficiency or deformity, &c. These changes, it has also been seen, are important or serious in proportion as they involve, *firstly*, contraction of one or more of the orifices and consequent obstruction to the free passage of blood; *secondly*, insufficiency of the valves or patency of one or more of the orifices, and consequent regurgitation; *thirdly*, contraction and patency combined. These immediate effects give rise to those secondary and remote derangements of the circulation which result from valvular lesions and eventuate in enlargement of the heart, the latter progressively increasing in proportion to the duration and amount of either obstruction or regurgitation, or both. But there are valvular lesions which involve neither contraction nor insufficiency, and, consequently, do not occasion an appreciable amount of either obstruction or regurgitation. Thickening, roughening, vegetations, &c., may be present without the results just mentioned, and therefore without the ulterior consequences in which consists the importance or seriousness of valvular lesions. Valvular lesions by no means necessarily involve any immediate danger; they may exist for a long period and no evils arise from them. In a practical point of view, this is a fact of great importance when it is considered that lesions which are innocuous, as well as those which are serious, give rise to endocardial murmur. It is to be borne in mind that valvular lesions which do not occasion any of the evil consequences arising from obstruction or regurgitation, nevertheless may involve the physical conditions requisite for the production of murmur. This important point will recur after the following questions have been considered: Are valvular lesions which do involve either obstruction or regurgitation, or both, uniformly or generally accompanied by murmur? What are the circumstances which, considered in connection with the presence of a murmur, enable the auscultator to determine whether existing lesions do involve either contraction or insufficiency, or both? Can the particular seat of valvular lesions be determined, and, if so, in what manner? The practical importance of these questions is sufficiently obvious. Before proceeding to their consideration, the reader may be reminded of the fact that in the vast majority of the cases of valvular lesions they are confined to the left side of the heart, affecting the aortic or mitral orifice, singly or combined. The questions just propounded, therefore, will relate mainly to the valves and orifices connected with the left ventricle.

Are valvular lesions which involve either obstruction or regurgitation, or both, uniformly or generally accompanied by murmur?

This question may be answered affirmatively as regards the *general*, but not as regards the *uniform* occurrence of murmur. Clinical experience shows that in the vast majority of cases murmur coexists; and it is probable that in most of the instances in which at a certain stage in the progress of the disease careful physical exploration fails in discovering any murmur, it either has existed or it becomes developed subsequently. Cases occur in which valvular lesions involving considerable and even great obstruction and regurgitation are found after death, and no murmur had been discovered during life. Such cases are, however, extremely rare, and, in general, it is fair to presume that murmurs had existed, and disappeared in the progress of the disease. Owing to the enfeebled action of the heart which often precedes, for a greater or less period, a fatal termination, a murmur which has existed may disappear, the blood not being propelled with force sufficient for its continuance, notwithstanding the persistence of physical conditions sufficient for its production. The practical bearing of the question under consideration relates chiefly to the value of the organic murmurs in a negative point of view; in other words, to the evidence afforded by the absence of murmur against the existence of valvular lesions. Clinical facts authorize the statement that, while the absence of murmur is not positive proof of the non-existence of serious valvular lesions, the probability of such lesions being present when a murmur is not discoverable is exceedingly small; so small, indeed, that it may be almost said to be with safety disregarded in diagnosis, especially if those cases are excluded in which an exploration of the chest is not made until the action of the heart is weakened by the failure of the vital forces, or by any causes depressing the muscular power of the organ. It is a point of great importance to determine in individual cases whether valvular lesions do, or do not, exist. The presence of a murmur by no means warrants the conclusion in all cases that lesions do exist, as will appear more fully after the inorganic murmurs have been considered. The absence of murmur, on the other hand, warrants the conclusion that lesions do not exist, the probability of error being exceedingly small, provided the heart be not from any cause greatly enfeebled.

What are the circumstances which, taken in connection with murmur, enable the auscultator to determine whether existing lesions involve either obstruction or regurgitation, or both?

With reference to this question, as well as to that which follows, it is necessary to have a clear idea of the relations of endocardial murmurs to the two sounds of the heart, respectively, and to the different currents of blood. After the systolic contraction of the ventricles, the blood passes through the auriculo-ventricular orifices from the auricles into the ventricles. Limiting the attention to the left side of the heart, the current of blood from the left auricle, through the mitral orifice, into the left ventricle, may be designated the *direct mitral current.* The systolic ventricular contractions impel the blood from the cavity of the ventricle into the aorta. The current of blood from the cavity of the left ventricle into the aorta, may be distinguished as the *direct aortic current.* These are the normal blood-currents. Others are incident to disease. If the mitral valves be insufficient, more or less of the blood contained in the cavity of the left ventricle is driven backward by the ventricular systole into the left auricle. Here then, is a regurgitant current which does not exist when the valves are sufficient. It may be called a *mitral regurgitant current.* If insufficiency of the aortic valves occur as an effect of lesions in this situation, a portion of the blood which remains in the aorta after the ventricular systole regurgitates into the ventricular cavity. This may be distinguished as an *aortic regurgitant current.* Now, each of these four currents may give rise to a murmur. Murmurs produced by these different currents may be named accordingly. Hence, there may be a *mitral direct murmur;* a *mitral regurgitant murmur;* an *aortic direct murmur*, and an *aortic regurgitant murmur.* These several murmurs sustain different relations to the heart-sounds, as will be obvious on a little consideration.

A *mitral direct murmur* begins after the diastolic or second sound of the heart, and precedes the systolic or first sound; in other words, it takes place during the long silence or pause which separates the diastolic and systolic sound. If we divide the murmurs into two classes (which it is convenient to do), namely, into diastolic and systolic murmurs, according to their relations, respectively, to the two heart-sounds, a mitral direct murmur will be included in the class of diastolic murmurs. To be more explicit, it occurs just before the systolic sound, and is, therefore, properly called a pre-systolic murmur. For convenience, however, it may be distinguished as the *mitral diastolic murmur.*

A *mitral regurgitant murmur*, on the other hand, being produced by the ventricular systole, commences with the systolic sound. It is, therefore, a systolic murmur, and may be called the *mitral systolic murmur.*

An *aortic direct murmur*, also produced by the ventricular systole, is a systolic murmur; it commences with the systolic sound, and may be called the *aortic systolic murmur.*

An *aortic regurgitant murmur*, on the other hand, produced by a retrograde current from the aorta into the ventricle after the systolic contraction, commences with the second or the diastolic sound. It is, therefore, a diastolic murmur, and may be called the *aortic diastolic murmur.*

The following recapitulation shows at a glance, the titles of the foregoing murmurs and their relations, respectively, to the blood-currents and heart-sounds:

Systolic murmurs commencing with the systolic or first sound of the heart, consist of, 1st. A mitral regurgitant or a mitral systolic murmur; and, 2d. An aortic direct or an aortic systolic murmur.

Diastolic murmurs commencing with or following the diastolic or second sound of the heart, consist of, 1st. A mitral direct or a mitral diastolic murmur; and, 2d. An aortic regurgitant or an aortic diastolic murmur. Each of these four organic murmurs claims distinct notice with reference to the important practical question under consideration.

1. *Mitral Direct or the Mitral Diastolic Murmur.*—This is less frequent in its occurrence than either of the other three murmurs. Some have disputed the possibility of this murmur on the ground that the contraction of the left auricle cannot impel the current of blood through the auriculo-ventricular orifice with force enough to give rise to an audible sound. Not only does it occur, but its occurrence is far less rare than has been generally supposed. Of 123 of my cases in which mitral lesions either existed alone, or were associated with aortic lesions, the mitral direct murmur is noted as present in 47. At one time, in a single medical division at Bellevue Hospital, there were five examples of the mitral direct murmur.

The mitral direct murmur generally denotes a particular kind of lesion, namely, union of the mitral curtains, leaving a slit-like and more or less contracted aperture between the auricle and ventricle. The quality of the murmur is almost always highly distinctive of it. It resembles the sound produced by throwing the lips or the tongue into vibration with the expired breath. It may be distinguished as a blubbering sound when this quality is strongly marked. It is a rough, not a soft, murmur. Its uniformity, in almost all the cases in which it is observed, is remarkable. Of all the cases in which I have noted its occurrence, in only one case was this characteristic quality wanting; in this single case it was a soft blowing murmur. The comparison of the murmur to the vibration of the lips or the tongue suggests the mechanism of its production. The mechanism is the same as in these illustrations. The current of blood, driven through the slit-like aperture, throws the adherent curtains into vibration precisely as the lips and tongue are made to vibrate with the expired breath. An examination of the heart *post-mortem*, in the cases in which this murmur was marked, shows flexibility of the curtains, in addition to contraction by the adherence together of their sides. The murmur is slight or wanting if the curtains be much thickened or made rigid by calcareous deposit.

The intensity of the murmur has seemed to give support to the opinion, held by some, that the force of the mitral direct current of blood is insufficient for its production. The intensity in some cases is notably great. With the explanation of its mechanism, however, which has been given, it is not difficult to understand that the murmur may be quite loud notwithstanding the auricular contraction is feeble in comparison with the systole of the ventricles. Let it be considered what a loud blubbering sound may be produced by the vibration of the lips or tongue with a feeble expiratory movement of the breath! The loudness of the murmur, other things being equal, will, of course, be proportionate to the force with which the auricle contracts; and, hence, hypertrophy of the muscular walls of the auricle will increase the loudness of the murmur. It is, however, intelligible that the current of the blood due to the elasticity of the auricle, before the auricular contraction begins, may have force enough to throw the curtains into vibration and produce an audible sound. This will meet another objection to the possibility of the production of this murmur by the mitral direct current, namely, that the duration of the murmur is sometimes longer than the duration of the muscular contraction of the auricle.

As regards the time when this murmur occurs, that is, its relation to the heart-sounds, it begins after the second or diastolic sound, and almost invariably it continues up to the occurrence of the succeeding first or systolic sound. Thus, although it occurs during the diastole, and is therefore in the class of diastolic murmurs, it is more closely related to the first than to the second sound. The term pre-systolic denotes the time of its occurrence. I have met with but a single case in which this murmur ended before the occurrence of the first sound. Generally it is more intense at its ending than at its beginning; and it seems to be suddenly, as it were, cut off with the occurrence of the first sound of the heart. Niemeyer quotes Traube as stating that if the action of the heart be much retarded by digitalis, this murmur sometimes ends before the first sound of the heart. The murmur is often supposed to be a mitral regurgitant murmur. It is generally considered to be a mitral regurgitant murmur by those who deny its existence. But after a careful study of its relation to the first sound of the heart, this error can hardly be committed. A mitral regurgitant murmur cannot begin prior to the ventricular systole, that is, before the first sound of the heart. But the mitral direct murmur always ends with the ventricular systole; the contraction of the ventricle, of course, must arrest instantly and completely the mitral direct current which causes the murmur. The mitral direct murmur ends, when the mitral regurgitant murmur, begins. A clinical illustration of the murmur is rendered more striking when it is associated with a mitral regurgitant murmur, than when it exists alone, if, as is usually the case, the two murmurs combined differ notably as regards quality. When the two murmurs exist together, the mitral direct, with its characteristic blubbering quality, continues until the first sound of the heart occurs; then begins the mitral regurgitant murmur, which is usually a soft murmur. The first sound of the heart, thus, is between the two murmurs, denoting the ending of the one and the beginning of the other.

Other points relating to the localization of this murmur by auscultation, will be considered in connection with the question which follows that under present consideration.

An important fact concerning the mitral direct murmur may be here stated. This murmur may be produced, and it may be quite intense, when the mitral valves are not the seat of any lesion. I have recorded three cases in which the murmur was loud, and the mitral valves were found *post-mortem* to be normal. In each of these cases there were aortic lesions rendering the aortic valves insufficient. The explanation of the occurrence of a mitral direct murmur, under these circumstances, involves a point connected with the physiological action of the auricular valves. Experiments show that when the ventricles are filled with a liquid, the valvular curtains are floated away from the ventricular walls, and coming into contact, close the auricular orifice. In fact, as was first shown by Baumgarten and Hamernik, of Germany, a forcible injection of liquid into the left ventricle through the auricular orifice will cause a complete closure of this opening by the coaptation of the mitral curtains, so that, as these authors contend, the natural closure of the auricular orifice is effected, not by the contraction of the ventricle, but by the forcible current of blood propelled into the ventricle by the auricle. However this may be, the fact that the mitral curtains are floated out and brought into apposition to each other by simply distending the ventricular cavity with liquid, is sufficiently established and easily verified. Now, in cases of considerable aortic insufficiency, the left ventricle is rapidly filled with blood flowing back from the aorta, as well as from the auricle before the auricular contraction takes place. The mitral curtains, under these circumstances, are brought into coaptation, and when the auricular contraction takes place, the mitral direct current passing between the curtains throws them into vibration, and gives rise to the characteristic blubbering murmur. The physical condition is in effect analogous to contraction of the mitral orifice from an adhesion of the curtains at their sides, the latter being the condition, as has been seen, which gives rise to the murmur in the great majority of cases.

Dr. W. T. Gairdner has proposed to distinguish this murmur by the name auricular systolic murmur.[1]

Mitral obstructive lesions increase the intensity of the pulmonary second sound of the heart. The significance of this sign has been alluded to in connection with the subject of hypertrophy of the right ventricle.[2] It will be noticed presently under the head of abnormal modifications of the heart-sounds incident to valvular lesions. Symptoms, in distinction from signs, are also to be taken into account. Those which, in connection with the presence of a mitral direct murmur, point to mitral contraction, and which afford, measurably, evidence of the amount of obstruction, are phenomena denoting congestion of the lungs, namely, dyspnœa, defective oxygenation of the blood, hæmoptysis, and pulmonary apoplexy. The pathological relations of these events have been already considered.[3]

2. *Mitral Regurgitant or Systolic Murmur.*—This is the murmur most frequently met with in cases of organic disease of the heart. Whenever the mitral valve is insufficient, a portion of the blood contained in the left ventricle is driven backward by the ventricular systole into the left auricle. This regurgitant cur-

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[1] For an argument against the existence of a mitral direct murmur, the reader is referred to a paper by Dr. J. R. Leaming, New York Med. Journal, June, 1868. Dr. Leaming attributes the production of the murmur to the vibration of the *chordæ tendineæ* occurring at the commencement of the ventricular systole. According to this view it is a systolic, not a pre-systolic murmur.

[2] Chap. I, page 67.

[3] *Vide* Chap. III.

rent passes through an orifice frequently contracted and the surfaces roughened by calcareous deposit, warty excrescences, &c. With a mitral regurgitant current, a murmur is almost invariably produced; the exceptions are so few that, practically, they may be almost disregarded. Is it true, on the other hand, that a systolic murmur referable to the mitral orifice as uniformly denotes the existence of insufficiency or regurgitation? This question must be answered in the negative. There are rules, to be presently considered, which enable the auscultator to localize a systolic murmur at the mitral orifice. Now, of the cases in which a murmur is referred to this situation, in only a certain proportion does regurgitation occur; in other words, a murmur may be produced at or near the mitral orifice, due to roughness, calcareous deposit, &c., without the valves being insufficient. It is important that this fact should be borne in mind. The gravity of valvular lesions, as has been seen, depends on the amount of obstruction and regurgitation resulting from them; hence, the importance of bearing in mind that a mitral systolic murmur is not always, strictly speaking, a regurgitant murmur, that is, the murmur may be produced without regurgitation. What, then, are the circumstances connected with the murmur which denote insufficiency or regurgitation? The diffusion of the murmur is an important point with reference to this question. If the murmur be diffused over the left side, extending laterally, and, as is not infrequently the case, to the posterior aspect of the chest, regurgitation may be assumed. This diffusion is more valuable in a positive than in a negative point of view; that is to say, regurgitation may exist without this diffusion of the murmur, whereas the converse, as a rule, does not hold good. In view of what has just been stated, instead of using the term mitral regurgitant murmur to denote a murmur at the mitral orifice, which may or may not, proceed from a mitral regurgitant current, it would be more correct to employ the term mitral systolic murmur, qualifying it by the words with regurgitation, or non-regurgitant, according to the presence or absence of the circumstances which show that the murmur denotes insufficiency of the mitral valves. Other associated circumstances pointing to regurgitation are the symptoms and pathological events proceeding from pulmonary congestion, which have been considered in the preceding chapter. Considerable regurgitation, however, may continue for a long time before these become developed. Clinical observation shows that mitral regurgitation may be long borne without serious inconvenience, especially if mitral contraction do not coexist. Symptoms, therefore, do not afford much aid in an early diagnosis of mitral insufficiency. A comparison of the aortic and the pulmonary second sound has an important bearing on the question just stated. Greater relative intensity of the pulmonic second sound is a sign of marked significance in this connection. It has a twofold significance. The intensity of the aortic second sound is diminished in proportion to the amount of blood which regurgitates through the mitral orifice, the stream which should pass into the aorta with each ventricular systole being lessened. The abnormal feebleness of the aortic second sound is thus proportionate to the degree of mitral insufficiency. In this point of view, the greater relative intensity of the pulmonic second sound is significant. But the intensity of this sound becomes actually increased. When this is the case, it shows that the regurgitation has induced hypertrophy of the right ventricle in the manner already described. Attention to the diffusion of the murmur, and a comparison of the aortic and the pulmonic second sound, enable the auscultator generally to determine whether a mitral systolic murmur denotes insufficiency, and also to form an idea of the amount of regurgitation.

Mitral insufficiency may exist either with or without mitral contraction, and, conversely, the latter may exist with or without the former. The lesions which occur in this situation, however, involve insufficiency without contraction oftener than contraction without insufficiency, but both are not infrequently combined. It follows from these facts that a mitral direct murmur and a mitral systolic murmur may exist either separately or conjointly; that the former existing alone is comparatively infrequent, while it is common to meet with the latter by itself, and that the instances in which both are conjoined are more frequent than the instances in which the first exists separately. Of 47 cases in which the presence of the mitral direct murmur was noted, it was accompanied by the mitral systolic murmur in 25, and the latter murmur was not associated in 22.

3. *Aortic Direct or Systolic Murmur.*—In frequency of occurrence, this ranks next to the mitral systolic murmur. Of 55 cases of valvular lesions in which either a mitral regurgitant or an aortic direct murmur existed separately, the former was

present in 38, and the latter in 21. This murmur, assuming, for the present, that it proceeds from organic lesions, denotes a serious affection, or otherwise, according to the effect of the lesions as regards obstruction at the aortic orifice. The physical conditions necessary for the production of a murmur in this situation may exist without any appreciable obstruction. Such instances are not rare. There may be, under these circumstances, no immediate danger and no troubles referable to the heart. The physical conditions giving rise to the murmur may remain for an indefinite period innocuous. On the other hand, in proportion as obstruction to the aortic blood-current is involved, evils ensue, namely, accumulation of blood in the ventricular cavity, enlargement of the left ventricle, and eventually, in some cases, enlargement of the left auricle, followed by pulmonary congestion, and the more remote consequences which are essentially those resulting from obstructive and regurgitant lesions at the mitral orifice. There are no constant characters pertaining to the murmur itself which enable the auscultator to determine whether the lesions do, or do not, involve obstruction. Marked roughness or a musical intonation renders probable contraction at the orifice, due to the expansion and rigidity of one or more of the valvular segments, or to the presence of an abundant morbid deposit. But these anatomical conditions may exist without either roughness of the murmur or a musical intonation; and, hence, the absence of the characters just named is not evidence against the existence of obstructive lesions. With reference to this point, it is important to compare the aortic with the pulmonic second sound of the heart. If the aortic sound retain its normal intensity, this shows that the aortic valves are competent to fulfil their function. In a large proportion of the cases of obstructive lesions at the aortic orifice, the valves are involved sufficiently to compromise, to a greater or less extent, their function, and impair the intensity of the aortic second sound. It is, however, to be stated that lesions may exist which involve obstruction without impairing the valves; in such cases, of course, the aortic second sound may have its normal intensity notwithstanding obstruction exists. Aside from attention to the aortic sound, the evidence of obstruction, and also of its degree and duration, must be derived from the amount of enlargement of the left ventricle and the remote effects of the heart affection. The cardiac enlargement, however, and the remote effects may proceed alike from aortic obstruction and aortic regurgitation singly or combined, as in cases of mitral lesions they result from either contraction or insufficiency. Enlargement of the heart accompanying valvular lesions, either at the mitral or aortic orifice, is, in general, proportionate to the amount and duration of obstruction or regurgitation, or both, which the lesions involve. The enlargement alone, therefore, does not enable the diagnostician to discriminate between the obstructive and regurgitant lesions.

4. *Aortic Regurgitant or Diastolic Murmur.*—This ranks next to a mitral direct murmur as regards infrequency. It is more frequently met with than the latter, but less frequently than the two other murmurs. The gravity of the lesions represented by this murmur depends on the extent of insufficiency or the amount of regurgitation. Comparison of the aortic with the pulmonic second sound of the heart is of importance with reference to this effect of lesions. Has the aortic second sound its normal intensity and its valvular quality unimpaired, the inference is that the valves are not to much extent insufficient. Is the sound weakened and its normal quality not distinctly defined, this is evidence that the amount of regurgitation is not insignificant. Is the sound extinguished, as is sometimes observed, either destruction or rigid expansion of the several valvular segments has taken place. The degree to which the sound is weakened and indistinct may be taken as, in a measure at least, a criterion of the amount of damage which the valves have received.

An aortic direct murmur and an aortic regurgitant murmur, may be present singly or combined. Regurgitation may take place without murmur, owing to the retrograde current being too feeble to occasion an audible sound. Absence of an aortic regurgitant murmur, therefore, is not positive proof that there is no regurgitation. In the majority of the cases in which lesions of the aortic valves give rise to a regurgitant murmur, a direct or systolic murmur coexists. The aortic direct and the aortic regurgitant murmur, consequently, are associated oftener than the latter is observed disconnected from the former.

It has been seen that a mitral systolic murmur does not always involve mitral regurgitation. In like manner, an aortic diastolic murmur may take place without aortic regurgitation.

Roughness of the inner surface of the aorta above the aortic valves may occasion a murmur with the retrograde movement of the column of blood within the vessel, although the aortic valves are sufficient. This retrograde movement is of little extent, and the murmur is correspondingly brief. The closure of the aortic valves instantly arrests the murmur, and it takes place between the first and second sound. In order to distinguish this from an aortic diastolic murmur with regurgitation, it might be called a pre-diastolic aortic murmur. Notwithstanding the exceedingly short space of time to which this murmur is limited, it may be distinctly appreciated. I have noted several examples. The following is an account of one of these, dated August, 1866: "In a patient under observation at Bellevue Hospital, there is a soft aortic direct murmur which is followed by a very short rough murmur ending abruptly with the second sound. The latter is pre-diastolic, and is regarded as produced by the recoil of the arterial coats without regurgitation. The presence of this murmur, as distinct from the aortic direct murmur, was verified by Dr. H. Smith and Dr. Henry F. Walker, of the hospital staff." February 23d, 1868, I have noted: "The same is observed in a patient now in Bellevue Hospital;" and a similar note was made with reference to another hospital case in September, 1868.

An aortic, non-regurgitant diastolic murmur is then characterized by its ending abruptly with the second sound of the heart; whereas, an aortic regurgitant murmur continues more or less after the second sound. I am not certain that this non-regurgitant murmur can be distinctly appreciated unless it follows an aortic direct murmur, and differ from the latter in pitch or quality. But, probably, an aortic direct murmur is always present under the anatomical conditions giving rise to the non-regurgitant diastolic murmur.

The organic murmurs which have been described are variously combined in different cases. The murmurs produced at each of the two orifices, namely, the mitral and the aortic orifice, are not infrequently presented in combination. The four murmurs are sometimes associated in the same case. A single mitral murmur may be associated with either or both of the aortic murmurs, and one aortic murmur may be associated with either or both of the mitral murmurs. It is conceivable, indeed, that eight distinct murmurs may be combined in the same case, tricuspid and pulmonic murmurs, corresponding to those produced in the left side of the heart, being present. We come now to the consideration of the means by which the different murmurs, respectively, are localized and discriminated from each other.

Can the particular seat of valvular lesions be determined, and, if so, in what manner?

The first part of this inquiry has been already answered. It has been stated that it is practicable, generally, to localize valvular lesions. The mode in which this may be done is now to be considered. To refer a murmur to a particular valve or orifice seems, to one practically unacquainted with the subject, a refinement in diagnosis not only difficult but invested with an air of mystery. The rules, however, are extremely simple; their application is by no means intricate, nor does it require the exercise of any extraordinary skill or tact. The points involved in determining the particular seat of lesions, relate: 1st. To their relations to the heart-sounds; 2d. To the different situations in which murmurs are found to be most intense, and the different directions in which they are farthest propagated; 3d. To the pitch and quality of the murmurs; and 4th. To the condition of the heart-sounds considered in connection with the murmurs. In treating of this branch of the subject, it will be convenient to consider the murmurs as embraced in two classes, viz., *systolic* and *diastolic*, that is, accompanying either the first or the second of the heart-sounds. Each of these two classes embraces an aortic and a mitral murmur.

Localization of the Systolic Murmurs.—In tracing an endocardial murmur to its source, the first point is to ascertain whether it be a systolic or a diastolic murmur. Generally this is unattended with difficulty; but it may be attended with some difficulty arising from the rapidity or the irregularity of the heart's action. If the heart-sounds recur with great frequency, the systolic and diastolic sounds are not readily distinguishable from each other. The two sounds may follow so quickly that the difference in duration between the two pauses or intervals is scarcely apparent. Moreover, under these circumstances, the first sound loses its distinctive characters as regards its relative length and quality, and the two sounds become very nearly identical. If the action of the heart be not notably rapid or irregular, attention to the rhythmical succession of the two sounds will enable the auscultator to determine which is the first and which is the second sound, aside from the characters distinguishing the one from the other in health. These characters cannot be relied upon, because, as will be seen presently, they are liable to be considerably impaired by disease, and in health they are marked only over the apex of the heart. Whenever there is doubt or difficulty in determining whether a murmur be systolic or diastolic, it is to be recollected that the first or systolic sound of the heart is synchronous with the apex-beat and the carotid pulse. In cases of great frequency or irregularity of the heart's action, therefore, the relation of murmurs to the heart-sounds may be determined by ascertaining their relation to the apex-beat or the carotid pulse. The latter is generally the most available. If the beginning of a murmur be coincident with the carotid pulse, it is either an aortic or a mitral systolic murmur.

In auscultating with reference to murmurs, it is to be borne in mind that, other things being equal, their intensity will be in proportion to the power of the heart's action. A murmur may at one time be inappreciable, owing to feebleness of the action of the heart, and, at another time, when, from any cause, the heart acts with more power, it may be distinct and even intense. This statement will apply to all the murmurs. Before deciding, therefore, on the absence of murmur, it is sometimes advisable to excite the heart's action by muscular exertion. For this purpose the patient may be requested to walk more or less rapidly several times around the room, and the stethoscope applied directly after this exercise.

In localizing the murmurs, as well as in discovering them when they are feeble, the auscultator who is accustomed to use the binaural stethoscope has a great advantage over those who employ other instruments or who rely upon immediate auscultation.

Assuming that a murmur has been ascertained to be systolic, the next question is, How is it to be traced to either the mitral or the aortic orifice? If it be a mitral systolic murmur, its maximum of intensity is at or near the apex of the heart. In some cases it is most intense at the point where the apex-beat is either seen, felt, or determined by auscultation; but in other cases the intensity is greatest at a little distance to the left of the apex-beat. When the latter is the case, the explanation is probably that given by Sibson, namely, the murmur is somewhat obscured directly over the apex by the intensity of the first sound, and sometimes by tinnitus.[1] This murmur may be confined within a circumscribed space around the apex. It is generally heard over the body of the heart, within the superficial cardiac region, but often with diminished intensity. Above the base of the heart it is often feeble or wanting. It is not propagated into the carotids. If it be transmitted to the upper part of the chest, as is sometimes observed when it is unusually loud, the intensity is less than over the body and apex of, or below the heart. It is often diffused over the left lateral aspect of the chest, and it may extend to the posterior aspect on the left, and sometimes on the right side. When heard on the back, its intensity is greater below than above the spinous ridge of the scapula, the maximum being near the lower angle of the scapula. I have met with cases in which it was heard in the lateral regions of the left side of the chest, and on the back at the lower angle of the scapula, and not heard over the apex. The quality of the murmur may be soft, rough, or musical. The pitch varies in different cases; in the larger number of cases it is represented by the whispered word WHO. The mitral valvular element of the first of the heart-sounds is fréquently diminished or wanting, leaving the element of impulsion unduly predominant or solely present.[2] The diminution of this element is in proportion to the injury of the valve which the lesions have occasioned, and its absence shows that the valve is nearly or quite useless. The aortic second sound is diminished in intensity in proportion to the amount of blood which regurgitates through the mitral orifice. The pulmonic second sound is thereby rendered relatively more intense, and its intensity is often actually increased by hypertrophy of the right ventricle.

The foregoing points distinguish a mitral regurgitant systolic murmur. If, on the other hand, a systolic murmur be an aortic direct murmur, its maximum of intensity is at or above the base of the heart. Its intensity is less over the body of the heart, within the superficial cardiac region, than at the base, and it may be lost in the former situation. It is still more feeble and is often lost over the apex; and it is very rarely propagated below this point. The particular situation where it is most intense, is usually in the second intercostal space nigh to the sternum. In the second intercostal space on the left side nigh to the sternum the intensity is, in general, less than at the corresponding point on the right side; but exceptionably the intensity is greater in the left second intercostal space. From the base of the heart it is propagated upward for a greater or less distance, usually more so on the right than on the left side. It is often pretty loud at the sternal notch. It is heard on the neck over the carotids. It is sometimes heard on the posterior surface of the chest, and when this is the case its maximum is in the left interscapular space on a level with the spinous ridge of the scapula. It is soon lost below this point. The murmur may be soft or rough, the latter quality being less frequent than the former. It is, however, oftener rough than a systolic mitral regurgitant murmur. The pitch is usually higher than in the majority of the instances of a mitral regurgitant murmur, often being represented by the letter R, whispered. The pitch, however, varies considerably in different cases. It has a musical intonation oftener than a mitral regurgitant murmur. The aortic second sound of the heart is frequently impaired and may be extinguished, the pulmonic second sound remaining.[1] The extent to which this sound is compromised, will, of course, correspond to the amount of injury to the aortic valves occasioned by the lesions which give rise to the murmur.

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[1] Medical Anatomy.

[2] For an account of the two elements of the first or systolic sound, and the manner of exploring for the mitral and tricuspid valvular elements of this sound, see Chapter I, page 61, *et seq.*

The two systolic murmurs may be associated in the same case. To determine the presence of both these murmurs, the incidental circumstances distinctive of each, should, in the first place, be sought after. If a murmur heard at the apex be transmitted over the left lateral aspect of the chest, and it be heard at the lower angle of the scapula behind, a mitral regurgitant murmur is present. If, now, a murmur heard at the base be heard over the carotids, there is also present an aortic direct murmur.

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[1] The situations in which the aortic and pulmonic second sounds may be studied separately, are the second intercostal spaces on the right and left side of the sternum. See Chapter I, page 60.

Again, if the two murmurs be present, the maximum of the intensity of one being at the base, and of the other at the apex, it will generally be found on passing the stethoscope slowly from the base to the apex, that the intensity of the murmur is notably lessened, or both murmurs may be lost, at an intermediate point over the body of the heart. A comparison of the murmur at the base and at the apex, as regards quality and pitch, will be likely to show the existence of the two murmurs. It will rarely happen that the two murmurs are identical in pitch and quality; one may be high and the other low in pitch, or one may be rough and the other soft. These points of difference establish the fact of duality. As a rule, these two murmurs in the same case may be localized with as much precision as when either is present without the other in different cases.

In determining that a murmur heard over the carotid is a transmitted aortic direct murmur, inasmuch as an inorganic murmur is frequently produced within the carotid, it is important to compare the murmur heard over the carotid with that heard at the base of the heart, as regards quality and pitch. If the carotid murmur be a transmitted aortic direct murmur, it will not differ notably in pitch and quality from the murmur at the base of the heart. It is to be added, moreover, that almost invariably an aortic direct murmur, however feeble it may be at the base of the heart, is transmitted into the carotids; and in some cases the murmur is louder over the carotids than at the base of the heart.

Localization of the Diastolic Murmurs.—A murmur having been ascertained to be diastolic, the question to be then settled is, whether it be a mitral direct murmur or an aortic regurgitant murmur.

A mitral direct murmur, as has been stated, is pre-systolic. It occurs just before the first or systolic sound, and is almost always continued up to that sound, owing to the fact that the contraction of the left auricle precedes, by a very short interval, the contraction of the left ventricle, the latter appearing to be a continuation of the former. None of the other murmurs occur in the same relation to the first sound of the heart, and hence, this alone is distinctive. Its maximum of intensity is within a circumscribed space around the apex of the heart. It is rarely diffused to much extent over the præcordia. The quality of the murmur is distinctive in the great majority of cases. The quality may be distinguished as vibratory, or, when strongly marked, blubbering, attributable, as has been stated, to the manner of its production. Commencing distinctly after the second sound of the heart, its intensity increases up to its becoming, as it were, merged into the first sound of the heart. Its abrupt ending with the first sound of the heart is usually a striking feature; it seems to be suddenly cut off, as indeed, the mitral direct current must be by the systole of the left ventricle.

The aortic second sound of the heart is weakened in proportion to the amount of mitral obstruction caused by the lesions which give rise to the murmur; and the pulmonic second sound becomes intensified when the lesions have led to hypertrophy of the right ventricle.

If the diastolic murmur be an aortic regurgitant murmur, it commences with, and follows the second sound of the heart. It is due to the same force which causes the aortic second sound, namely, the recoil of the elastic coat of the aorta, while the mitral direct murmur is produced by the contraction of the left auricle. The former occurs prior to the latter, and hence the commencement of the aortic regurgitant murmur takes precedence in point of time. The intensity of this murmur is usually greatest in the third or fourth intercostal space, nigh to the left margin of the sternum. As a rule it is feeble and soft. There are, however, exceptions to this rule; the murmur is sometimes quite intense, and it may be notably rough or musical. The pitch is usually low, being represented by the whispered word *awe;* but like the other murmurs the pitch is variable. The aortic regurgitant murmur is transmitted in a downward direction toward the epigastrium. I have known it to be appreciable as low as the umbilicus. It is generally heard at the base of the heart, that is in the second intercostal space, and it is usually louder on the right than on the left side of this space; but it is sometimes heard in the third or fourth intercostal space on the left side, when it is not appreciable at the base. Exceptionally, it is sometimes well marked over the apex of the heart when it is feeble or wanting at the base. It is frequently, but by no means invariably, heard over the carotids. Its intensity is greatest at its commencement, diminishing until the murmur becomes inappreciable.

As no other of the four murmurs under consideration commences with the second sound of the heart, it suffices for its recognition to make out this point; and if it be difficult to determine which of the heart-sounds is the first, and which the second, sound, the relation of the murmur to the second sound is shown by the interval between the murmur and the carotid pulse.

Frequently the two aortic murmurs are associated, one murmur then accompanies the first, and another murmur the second sound of the heart; or two murmurs at the base occur in the same rhythmical succession as the two heart-sounds. But it is sometimes the case that, when the two murmurs are present, they are not both heard at the base of the heart. The aortic direct may be heard at the base, and the aortic regurgitant may be heard below the base, namely, in the third or fourth intercostal space on the left side nigh to the sternum. As already stated, an aortic diastolic murmur may exist without regurgitation. The characters distinctive of such a murmur have been already sufficiently considered.[1]

The two diastolic murmurs may be associated. When this is the case, they are readily enough distinguished from each other. The aortic regurgitant murmur begins *with* the second sound, and, being more or less prolonged in the interval between the heart-sounds, becomes gradually inaudible. The mitral direct murmur begins *after* the second sound, and continues into the first sound. The aortic regurgitant murmur is generally soft; the mitral direct generally has a characteristic rough quality. The aortic regurgitant murmur has, in most cases, its maximum of intensity at or near the base of the heart; the mitral direct murmur, however intense, is almost always limited to a circumscribed space around the apex of the heart.

If the aortic lesions occasion much damage to the valves, the aortic second sound of the heart will be proportionately weakened.

It is hardly necessary to state that in localizing three or all four of the organic murmurs emanating from the left side of the heart, the rules for distinguishing them separately, or in pairs, which have just been considered, are to be applied. To assist the reader in fixing in the memory the points involved in the localization of each of these murmurs, the following summary is subjoined:

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[1] *Vide* page 212.

RECAPITULATION OF POINTS INVOLVED IN THE LOCALIZATION OF THE SYSTOLIC AND DIASTOLIC MURMURS REFERABLE TO THE MITRAL AND THE AORTIC ORIFICE.

SYSTOLIC MURMURS.

MITRAL REGURGITANT.	AORTIC DIRECT.
Maximum of intensity at or near the apex of the heart. Comparatively feeble or wanting at the base.	Maximum of intensity at the base of the heart in the second intercostal space, near the sternum. Intensity diminished over body of heart and at the apex.
Not propagated above the base of the heart. Not heard over carotids.	Propagated above the base of the heart, and generally heard over carotids.
Often diffused over left lateral surface of chest.	Rarely heard over left lateral surface of chest.
If heard in the interscapular space, most intense near the lower angle of scapula.	If heard in the interscapular space, most intense as high as the spinous ridge of scapula.
Aortic second sound weakened in proportion to the amount of regurgitation, but distinct.	Aortic second sound often weakened, and more or less indistinct.
Pulmonic second sound often intensified.	Pulmonic second sound less frequently intensified.
Mitral valvular element of the first sound more or less impaired.	Mitral valvular element of the first sound not impaired.

DIASTOLIC MURMURS.

MITRAL DIRECT.	AORTIC REGURGITANT.
Occurs just before the systolic or first sound, and ends with the occurrence of this sound. Usually vibratory or blubbering in quality.	Commences with and follows the diastolic or second sound. The quality usually soft.
Maximum of intensity over apex of heart.	Maximum of intensity over body of heart, near the sternum.
Generally not appreciable at the base of the heart.	Generally appreciable at the base of the heart.
Mitral valvular element of first sound impaired.	Mitral valvular element of first sound sound not impaired.
Pulmonic second sound often intensified.	Pulmonic second sound less frequently intensified.

PATHOLOGICAL IMPORT OF ORGANIC ENDOCARDIAL MURMURS.

It is highly important to form a just notion of the extent to which organic murmurs are available in furnishing information respecting pathological conditions. The considerations which have been presented have related mainly to diagnosis. They show that organic lesions are accompanied by an organic murmur in the great majority of cases; and, conversely, the absence of murmur renders it almost certain that organic lesions do not exist. So far, the practical value of auscultation in this application is very great. Further than this, valvular lesions may generally be localized at one or more of the orifices of the heart by attention to certain points pertaining to the murmurs. Moreover, the study of the murmurs enables the auscultator often to determine whether obstruction or regurgitation, or both, at one or more of the orifices, are consequent on existing lesions. These ends are sufficiently important to render invaluable the aid thus derived from auscultation. The information, however, derived from the murmurs is limited mainly to these ends. The character of the structural changes which have taken place, and the amount of damage which they have occasioned, are to be determined by other means than the study of the murmurs. In a practical view, it is far more important to establish these points than the mere existence of organic lesions of some kind, or their particular situation and their immediate effects on the blood-currents. In determining these points, the heart-sounds, in the first place, are to be studied with reference to abnormal modifications, or otherwise, and, in the second place, the existence and the extent of enlargement of the heart are to be ascertained. The means of ascertaining the existence of cardiac enlargement and measuring its extent have been fully considered in a former chapter. Abnormal modifications of the heart-sounds, in connection with endocardial murmurs, have also been referred to. The latter will be again noticed presently under a distinct head.

It is to be borne in mind that lesions which are innocuous as regards any immediate effects, that is, which occasion neither obstruction nor regurgitation, may give rise to murmurs. Serious lesions are by no means to be predicated on the existence of a murmur which is undoubtedly of organic origin. This is a practical precept to be enforced. Nor is the intensity of a murmur to be taken as any criterion of the importance of the lesions which give rise to it. An intense murmur may accompany trivial lesions, and, on the other hand, the most serious lesions may give rise to a feeble murmur. Indeed, feebleness of the murmur may constitute evidence of the gravity of lesions, showing that the heart has become weakened by the dilatation and over-distension of its cavity incident to the lesions. Roughness of a murmur also, it is to be recollected, is no guide to the nature or extent of the structural changes which it indicates. The same remark is applicable to the diffusion of a murmur, when it is aortic. Diffusion of a mitral regurgitant murmur, on the other hand, without the præcordial region, may, in general, be considered as denoting regurgitation; but it is in nowise a criterion of the amount of regurgitation, or, in other words, of the valvular insufficiency.

An organic murmur is in some cases neither propagated above nor in any direction beyond the heart, but about equally diffused within the limits of the præcordial region. The lesions giving rise to the murmur in these cases are generally within the left ventricle, and they may be situated either on the ventricular aspect of either the mitral or the aortic valves. It is difficult or impossible to localize the lesions under these circumstances. For an example, in a case in which vegetations of considerable size were dependent from the inferior surface of the aortic valves, the valves not being otherwise affected, the murmur was confined to the region of superficial cardiac dulness, its maximum of intensity not being marked at any point within this space. Murmurs of this description may be distinguished as *intraventricular murmurs*,[1] and the inference to be drawn from them is that the valves are not affected to the extent of interrupting materially their functions.

A systolic murmur either limited to, or having its maximum of intensity at or near the apex of the heart is sometimes observed, and after death nothing is found to account for it—the mitral valves appear to be normal. A number of such cases have been reported. Bristowe has published an account of six cases.[2] I have reported three cases in an article on the cardiac murmurs, published in the *American Journal of the Medical Sciences*, in 1852, and I have noted three additional cases since the publication of that article. It is possible, as will be seen presently, that such a murmur may occur in consequence of a functional incapacity of the mitral valves, due to spasm of the papillary muscles; but, in the six cases which I have observed, the murmur was too constant and too persistent for this explanation to be admissible. Such cases admit of different explanations. In some there may have been insufficiency of the mitral valves from enlargement of the mitral orifice, the existence of such a condition being overlooked. This explanation will only apply to cases in which the left ventricle is dilated. Bristowe supposes that, in some of these cases the murmur may be due to a "disproportion between the size of the ventricular cavity and the length of the chordæ tendineæ and musculi papillares. He attributes this disproportion to dilatation of the ventricle. The explanation will apply to cases in which the mitral orifice is not enlarged. An explanation offered by Dr. Hare is, that the murmur may be caused by a "lateral displacement of the origins of the musculi papillares in consequence of the rounded form which dilatation imparts to the heart."

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[1] *Interventricular*, in distinction from murmurs produced at the orifices and propagated for a greater or less distance beyond the heart, either above the base, if the lesions be aortic, or to the left of the heart if the lesions be mitral and involve insufficiency.

[2] Brit. & For. Med.-Chir. Review, July, 1861.

An explanation which I suspect will account for many of these cases, and which I believe will apply to all of my six cases, save one, is that the murmur is a tricuspid, and not a mitral, systolic murmur. In two of my cases this belief is supported by the fact that emphysema and notable cyanosis existed. In one of these two cases the murmur was not only loud but rough. In that case the walls of the right were nearly as thick as those of the left ventricle. There is another explanation which has been suggested to me, and I know not to whom belongs the credit for originating it. This explanation is, that the apex of the heart, in its systolic movements, presses the tongue of lung which is apt to project over it, against the thoracic walls and expels the air from the pulmonary cells with sufficient force to produce a blowing sound. In one of my cases this explanation seemed admissible, in view of none of the others being available, and of the fact that the murmur was heard chiefly at the end of the inspiratory act.

A murmur emanating from the left ventricle, and liable to be considered as a mitral systolic murmur, may be due to perforation of the interventricular septum. The perforation may be congenital and due to arrest of development, or it may be the result of disease. An example of the latter has fallen under my observation, the case not being included in the six cases just referred to. A condensed account of this case is as follows:

The patient, a young man, aged about 25, had always had good health prior to June, 1862. He then received a severe blow from the end of a board in a saw-mill, directly upon the præcordia. There was no penetration, but only a contusion. Dr. Henry Van Blarcom, of Paterson, N. J., saw him shortly after the injury, and, at that time, on a careful examination, discovered no cardiac murmur. The patient kept about, but he was feeble and suffered from dyspnœa. Not long after the injury, the precise time not being noted, Dr. Van Blarcom found a loud endocardial murmur. About six weeks after the accident, the patient had general dropsy, which had continued, varying in amount, up to the time when I saw him in May, 1863. There was then moderate anasarca, together with a very large collection of liquid in the peritoneal sac. On physical examination, the heart was but little, if at all, enlarged ; but there was a very loud and slightly rough systolic murmur at the apex, together with well-marked purring tremor. The veins of the neck and upper extremities were much enlarged. The murmur was supposed to be a mitral systolic murmur.

The patient was much relieved by tapping the abdomen, and this operation was subsequently repeated several times. Death occurred in March, 1864.

On examination after death, the heart was moderately enlarged, the left ventricle being dilated. All the valves were sound and apparently sufficient. A fistulous orifice existed at the lower part of the interventricular septum, large enough to admit the end of the forefinger. Around the perforation, within the left ventricle, there was a patch of opacity as large as a half dollar. This opacity was due to exudation beneath the endocardium. Also, within a circumscribed space around the perforation the muscular structure on section was found to be infiltrated with exudation matter, and the walls within this space were thinned. These appearances were considered as showing myocarditis, to which the perforation was attributable. In the right ventricle, at a point opposite to the perforation, there was an opaque patch of the size of a half dollar, attributable to the action of the current of blood driven through the orifice by the systole of the left ventricle. The situation of the perforation was such, that, during the diastole of the ventricle, blood must have flowed freely from the right into the left ventricular cavity ; hence, the dilatation of the left ventricle.

In treating of the subject of endocardial organic murmurs in this chapter, reference has been had, for the most part, to those occurring in connection with chronic valvular affections of the heart. Organic murmur, as will be seen hereafter, also occurs in connection with heart-clots, in cases of congenital malformations, and its development becomes an important physical sign of endocarditis.

INORGANIC MURMURS.

As already defined, a murmur is inorganic when it is produced independently of organic or structural lesions. An endocardial murmur may be present when there are no lesions. The practical importance of being able to determine whether an existing murmur be organic or inorganic is sufficiently obvious. This discrimination, happily, can be made in practice in the great majority of cases. The points involved in the discrimination claim attention in this connection.

An inorganic murmur, in most cases, proceeds from an abnormal change in the composition and properties of the blood. The precise nature of the change is perhaps not positively ascertained. At all events, a discussion of this subject need not be here introduced. Whatever be the requisite conditions, they occur in a certain proportion of cases of anæmia and chlorosis. The murmur in these instances is said to be of *hæmic* origin. It was observed by Marshall Hall, in his researches on the effects of the loss of blood, that the sudden abstraction of a large quantity of blood led to the development of a transient bellows murmur. Other observers have verified this fact. It is occasionally observed under circumstances which seem to render probable the supposition that it may proceed from deficient or irregular contraction of the papillary muscles, involving temporary insufficiency and regurgitation. Its occurrence in some cases of chorea has been accounted for in this way. Thus produced, the murmur is said to be of dynamic origin. It is produced in some persons in health by the violent action of the heart which follows active muscular exertion, disappearing when the organ resumes its usual tranquillity. It is occasionally observed in the course of a variety of affections, when its disappearance, leaving no signs or symptoms of cardiac disease, after recovery, shows that it does not proceed from organic causes. Acute articular rheumatism, the continued and eruptive fevers, uræmia, and hysteria, are among the affections in which it sometimes occurs. Its occurrence is not infrequent during pregnancy. What are the characters which distinguish these murmurs from those of organic origin?

Inorganic murmurs are uniformly systolic, *i. e.*, they accompany only the first of the heart-sounds. Diastolic murmurs are always of organic origin.

In the vast majority of cases, inorganic murmurs are heard at the base of the heart, and are not heard at the apex. This, at least, is true, as a rule, of cases of inorganic murmurs of *hæmic* origin. It is only the very rare and somewhat dubious instances of murmurs of *dynamic* origin, that are produced at the auriculo-ventricular orifices, and, consequently, heard at the apex. These are characterized by temporary duration or intermittency.[1] As a rule, organic murmurs are constant and persistent, whereas inorganic murmurs are fluctuating and variable, being sometimes discoverable only when the body is in a certain position.

An inorganic murmur is generally soft. Roughness, therefore, may be considered as favoring the conclusion that the murmur is organic. This statement will apply equally to an endocardial musical murmur. Exceptionally, however, an inorganic murmur is notably rough. The following is an example which has fallen under my observation: I visited in May, 1860, a female patient, who had a loud, rasping murmur which had led to the suspicion of aneurism. The patient was exceedingly anæmic; there was total loss of appetite, with vomiting and diarrhœa. The anæmia could not be accounted for; the case belonged among the cases described by Addison under the name idiopathic anæmia. The loud, rasping murmur was at the base of the heart on the right of the sternum, and a similar murmur was heard over the subclavian and carotid artery. On examination after death, in this case, the heart was found to be perfectly normal; the aortic orifice, the aorta, the subclavians and carotids, were free from any morbid change, and the lungs were healthy. The murmur was evidently due to a blood change.

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[1] For a consideration of an inorganic murmur heard over the apex or body of the left ventricle by Dr. D. M. Da Costa, *vide* Am. Journ. of Med. Sciences, No. for July, 1869.

An inorganic murmur may be produced either at the aortic or pulmonic orifice, or simultaneously at both orifices. If a murmur be pulmonic, as is rendered probable by its being either limited to, or having its maximum of intensity in, the left second intercostal space, it is probably inorganic, in view of the great infrequency of lesions situated at this orifice. Congenital malformations are to be excluded from this statement, for these are more liable to affect the pulmonic than the aortic orifice. In this connection it may be mentioned that pressure with the stethoscope in this intercostal space over the pulmonic artery will sometimes develop a bellows murmur in that vessel. This is observed in young persons whose costal cartilages are flexible. The murmur is due to pressure on the artery, as in the case of other arteries, more accessible, such as the carotid, iliac, femoral, &c. It is well known that light pressure on these arteries frequently develops a bellows murmur. Judging from my own observations, I should say that an inorganic murmur as often, if not oftener, emanates from the pulmonic as from the aortic orifice. An inorganic murmur in both these orifices may be inferred when the murmur is heard in the right and in the left second intercostal space, the pitch on the two sides differing so much as to show that there are two murmurs instead of a single murmur.

Inorganic murmurs occur in anæmic persons, and the palpable indications of anæmia are generally manifest. The coexistence of anæmia is a point to be considered in the discrimination. This condition, it is true, may coexist with valvular lesions, and it contributes to render more intense and diffused the murmurs due to the latter. Anæmia alone by no means warrants a conclusion that a murmur is inorganic, but, added to other evidence, it strengthens this conclusion.

Concurrent bellows murmurs emanating from the large arterial trunks, the subclavian, carotids, &c., not due to pressure with the stethoscope, are evidence that an endocardial murmur is inorganic. This evidence is by no means complete in itself, but adds weight to that derived from other sources. A continuous murmur or hum produced in the cervical veins is very generally, if not invariably, associated with an endocardial murmur of hæmic origin. This venous hum, called, after Bouillaud, by the French writers, *bruit de diable* (from its resemblance to the sound of the humming-top, which is known popularly in France as *le diable*), has heretofore given rise to considerable discussion as regards its source. Laennec, who first observed it, referred it to the arteries. In this he was, until lately, followed by most French writers. Its origin in the veins was first demonstrated by Dr. Ogier Ward. It is a sufficient demonstration of the correctness of the latter view that the murmur is suspended by interrupting the circulation through the veins, the arterial circulation continuing. The murmur is a continuous humming sound, having frequently a musical intonation. It is best heard just above the right clavicle, the patient being in the sitting or standing posture. The head of the patient should be turned as far as possible to the left, and the stethoscope placed on the right side, within the triangular space posterior to the sterno-cleido-mastoid muscle. The stethoscope should be shifted from point to point within this triangle, and the application made with varying degrees of pressure. These rules are important. By observing them a venous hum is a reliable sign of the conditions of the blood which give rise to inorganic murmur at the aortic or pulmonic orifice and within the larger arteries. So constant is the sign, that its absence is strong proof against the inorganic character of murmur at the base of the heart. Frequently, in conjunction with the venous hum in the neck, a systolic blowing is heard to emanate from the carotid artery. The venous hum is a valuable sign denoting the existence of anæmia when the anæmic appearance is not distinct; and, also, as evidence of the continuance of the anæmia, the disappearance of the sign showing that the anæmic condition has been removed.

Inorganic endocardial murmurs are much oftener observed in females than in males, a fact probably due to the greater frequency of anæmia in the former. Sex, therefore, is entitled to some weight in determining whether a murmur be organic or inorganic.

The heart-sounds, in connection with inorganic murmurs, retain their normal intensity and characters, or, if affected at all, their intensity is augmented; whereas, in connection with organic murmurs, they often present abnormal modifications which are to be presently considered.

Finally, organic murmurs, in the great majority of the cases of chronic disease, when these first come under the cognizance of the physician, are associated with more or less enlargement respecting pathological conditions. The considerations which have been presented have related mainly to diagnosis. They show that organic lesions are accompanied by an organic murmur in the great majority of cases; and, conversely, the absence of murmur renders it almost certain that organic lesions do not exist. So far, the practical value of auscultation in this application is very great. Further than this, valvular lesions may generally be localized at one or more of the orifices of the heart by attention to certain points pertaining to the murmurs. Moreover, the study of the murmurs enables the auscultator often to determine whether obstruction or regurgitation, or both, at one or more of the orifices, are consequent on existing lesions. These ends are sufficiently important to render invaluable the aid thus derived from auscultation. The information, however, derived from the murmurs is limited mainly to these ends. The character of the structural changes which have taken place, and the amount of damage which they have occasioned, are to be determined by other means than the study of the murmurs. In a practical view, it is far more important to establish these points than the mere existence of organic lesions of some kind, or their particular situation and their immediate effects on the blood-currents. In determining these points, the heart-sounds, in the first place, are to be studied with reference to abnormal modifications, or otherwise, and, in the second place, the existence and the extent of enlargement of the heart are to be ascertained. The means of ascertaining the existence of cardiac enlargement and measuring its extent have been fully considered in a former chapter. Abnormal modifications of the heart-sounds, in connection with endocardial murmurs, have also been referred to. The latter will be again noticed presently under a distinct head.

It is to be borne in mind that lesions which are innocuous as regards any immediate effects, that is, which occasion neither obstruction nor regurgitation, may give rise to murmurs. Serious lesions are by no means to be predicated on the existence of a murmur which is undoubtedly of organic origin. This is a practical precept to be enforced. Nor is the intensity of a murmur to be taken as any criterion of the importance of the lesions which give rise to it. An intense murmur may accompany trivial lesions, and, on the other hand, the most serious lesions may give rise to a feeble murmur. Indeed, feebleness of the murmur may constitute evidence of the gravity of lesions, showing that the heart has become weakened by the dilatation and over-distension of its cavity incident to the lesions. Roughness of a murmur also, it is to be recollected, is no guide to the nature or extent of the structural changes which it indicates. The same remark is applicable to the diffusion of a murmur, when it is aortic. Diffusion of a mitral regurgitant murmur, on the other hand, without the præcordial region, may, in general, be considered as denoting regurgitation; but it is in nowise a criterion of the amount of regurgitation, or, in other words, of the valvular insufficiency.

An organic murmur is in some cases neither propagated above nor in any direction beyond the heart, but about equally diffused within the limits of the præcordial region. The lesions giving rise to the murmur in these cases are generally within the left ventricle, and they may be situated either on the ventricular aspect of either the mitral or the aortic valves. It is difficult or impossible to localize the lesions under these circumstances. For an example, in a case in which vegetations of considerable size were dependent from the inferior surface of the aortic valves, the valves not being otherwise affected, the murmur was confined to the region of superficial cardiac dulness, its maximum of intensity not being marked at any point within this space. Murmurs of this description may be distinguished as *intraventricular murmurs*,[1] and the inference to be drawn from them is that the valves are not affected to the extent of interrupting materially their functions.

A systolic murmur either limited to, or having its maximum of intensity at or near the apex of the heart is sometimes observed, and after death nothing is found to account for it—the mitral valves appear to be normal. A number of such cases have been reported. Bristowe has published an account of six cases.[2] I have reported three cases in an article on the cardiac murmurs, published in the *American Journal of the Medical Sciences*, in 1852, and I have noted three additional cases since the publication of that article. It is possible, as will be seen presently, that such a murmur may occur in consequence of a guished, owing to the extent of injury to the valves, the tricuspid valvular sound may generally be distinguished by applying the stethoscope at or a little without the inferior or right border of the heart. Mitral regurgitation, in fact, leads to augmented intensity of the tricuspid sound by inducing hypertrophy of the right ventricle in the manner already described. It is to be borne in mind, that both the mitral and tricuspid valvular sound are lessened in intensity in proportion to the accumulation of blood within the ventricles at the time of the ventricular contractions. The explanation of this fact has been already given.[1] The accumulation of blood within the ventricles, other things being equal, will be proportionate to their weakness from dilatation or other causes. Aortic obstructive and regurgitant lesions also lead to over accumulation of blood in the left ventricle, and consequently to impairment of the intensity of the mitral element of the first sound. Mitral obstructive and regurgitant lesions, on the other hand, lead to over accumulation in the right ventricle, and consequently impair the intensity of the tricuspid element of the first sound.

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[1] *Interventricular*, in distinction from murmurs produced at the orifices and propagated for a greater or less distance beyond the heart, either above the base, if the lesions be aortic, or to the left of the heart if the lesions be mitral and involve insufficiency.

[2] Brit. & For. Med.-Chir. Review, July, 1861.

Mitral lesions involving obstruction or regurgitation, more especially the former, lead to diminished intensity of the aortic second sound, and increased intensity of the pulmonic second sound. The diminished intensity of the aortic sound is due to the column of blood propelled through the aorta by the ventricular contraction being lessened, either by the deduction of the quantity of blood which regurgitates, or by the deficient supply from the auricle to the ventricle. The increased intensity of the pulmonic sound proceeds from hypertrophy of the right ventricle. Both effects combine to render the greater intensity of the pulmonic, as compared with the aortic, second sound, a valuable sign of mitral obstruction or regurgitation, as was first pointed out by Skoda.

Aortic lesions affect the aortic second sound, other things being equal, in proportion to the extent of injury of the valves of the aorta. If the play of these valves be defective, the sound loses more or less of its normal intensity. The sound is extinguished when the valves are rendered useless or destroyed by disease. Instances of extinction of the aortic second sound are not very infrequent. In such instances the continuance of the pulmonic second sound shows that the loss of the aortic sound is not due to weakened action of the heart. Either an aortic direct or an aortic regurgitant murmur, or both, are present, indicating the fact of aortic disease, but the intensity and quality of the murmurs here, as in mitral lesions, do not constitute any criterion of the amount of damage to the valves. The abnormal modifications of aortic sound, however, afford definite information with respect to that important point.[1]

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[1] *Vide* page 86.

In determining if the relative intensity of the aortic and pulmonic second sound be normal or abnormal, it is to be borne in mind that it may be affected by morbid conditions extrinsic to the heart. Thus, tuberculous solidification of the lung in proximity to either the aorta or the pulmonary artery at the second intercostal space, by a better conduction of the valvular sound will render it apparently more intense. So, the anatomical relations of the aorta and pulmonary artery to the second intercostal space will be changed by contraction of the walls of the chest from tuberculous disease or after chronic pleurisy. Deformity of the chest from any cause may prevent a comparison of the aortic and pulmonic second sound. In certain cases of enlargement of the heart, the anatomical relations of the aorta and pulmonary artery may be altered so that the rules which have been stated are thereby rendered inoperative.

Purring Tremor.—This term is applied to a sense of vibration or thrill felt on placing the fingers or the hand on the præcordia. It is synonymous with the name applied to it by Laennec, viz., *frémissement cataire*, so called because it resembles the sensation communicated to the hand by the purring of a cat. Bouillaud compares it to the sensation felt when the hand is applied over the larynx of a person singing. These comparisons cause it to be easily recognized when met with for the first time. It is doubtless due to tremulous movements of the heart, which are propagated to the portion of the thoracic walls with which the heart is in contact.

Well-marked purring tremor may be considered as a sign denoting valvular lesions associated with hypertrophic enlargement of the left ventricle. If it occur under other circumstances, the instances are so infrequent that, for all practical purposes, the rule may be taken as invariable. The valvular lesions which most frequently give rise to it are those of the mitral orifice permitting free regurgitation. A regurgitant current driven through this orifice with an abnormal force, in consequence of the augmented muscular power of the ventricle, appears to be the immediate cause in the majority of instances. It accompanies or follows the ventricular systole, and is therefore synchronous with the first sound, the apex-beat and the pulse. A diastolic tremor must be exceedingly rare, but is stated by some clinical observers to occur occasionally. When due to mitral lesions, the tremor is felt within the superficial cardiac region below the level of the nipple. It may be more or less marked, the intensity depending, in a great measure, on the power with which the left ventricle contracts. It may be present or strongly marked when the action of the heart is excited by any cause, and absent or comparatively feeble when the organ is tranquil. In the progress of disease, it diminishes and ceases as the heart becomes weakened. Although it has a pathognomonic significance when present, its absence is in nowise evidence against the existence of organic lesions, for it is wanting in a large proportion of the cases in which lesions exist.

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[1] For a fuller consideration of this subject than is contained in this work, the reader is referred to the essay, by the writer, contained in the Transactions of the American Medical Association, vol. xi, p. 805.

The sign does not belong exclusively to mitral lesions. It accompanies, in some instances, aortic lesions associated with hypertrophy of the left ventricle. It is then felt nearer the base of the organ. A thrill due to the current of blood in the aorta is sometimes perceived above the heart in the second intercostal space on the right side. Vascular thrill in the carotids is sometimes marked in cases of aortic regurgitation; it occurs, also, in cases of anæmia without disease of the heart. With the aneurismal thrill all observers are familiar.

Cardiac tremor is not a sign of much practical value in view of the fact that it is present only in a small proportion of cases of valvular lesions, and since other physical signs, which are constant, are readily available for diagnosis. It is, however, of sufficient interest and importance to be kept in mind in exploring the chest for the physical evidence of cardiac disease.

Purring tremor is to be distinguished from the tactile fremitus incident to the presence of solid deposit on the pericardial surfaces. The latter, which will be noticed in connection with pericarditis, is always accompanied by an exocardial murmur or friction-sound on auscultation. Purring tremor, on the other hand, is associated with one or more endocardial murmurs.

DIAGNOSTIC CHARACTERS OF LESIONS AFFECTING THE MITRAL, AORTIC, TRICUSPID, AND PULMONIC VALVES OR ORIFICES.

The diagnosis of valvular lesions is based on physical signs, together with their symptoms and pathological effects. These have been considered. The significance and diagnostic value of the different signs, symptoms, and effects, have been pointed out. It remains to group together the more important of the characters which pertain to the different lesions respectively. This will be done as concisely as possible, the object being to present a brief summary of the distinctive features belonging to each of the several classes of lesions. Lesions affecting the different valves or orifices will now be taken separately as points of departure, viz., mitral, aortic, tricuspid, and pulmonic lesions. These will be considered under distinct heads. The signs, symptoms, and effects of tricuspid and pulmonic lesions have thus far been passed by, owing to the comparative infrequency of their occurrence, exclusive of congenital malformations, and in order not to render the subject needlessly complicated to the student. It will suffice to present briefly the characters which belong to these lesions in this division of the subject. As involved in congenital malformations, they will be referred to hereafter.

DIAGNOSTIC CHARACTERS OF MITRAL LESIONS.

Physical Signs.—An endocardial systolic murmur is present in the majority of cases, with the traits which distinguish a mitral regurgitant murmur, viz., its maximum of intensity at or near the apex of the heart, the intensity diminishing as the stethoscope is carried upwards over the body of the heart; generally feeble or lost above the base of the organ; not propagated into the carotids; often diffused over the left lateral surface of the chest, and not infrequently heard on the posterior surface, at the lower angle of the scapula, and in the interscapular space below the level of the spinous ridge of the scapula; the murmur more or less intense; generally soft, but sometimes rough, and occasionally musical.

The mitral portion of the valvular elements of the first sound of the heart is often more or less impaired, or extinguished, the tricuspid portion of the same element remaining distinct, or abnormally intense. The aortic second sound is weakened; the pulmonic second sound is often intensified. Enlargement of the heart exists in the majority of the cases which come under observation. These are the signs of mitral valvular lesions involving insufficiency or regurgitation through the mitral orifice, that is, mitral regurgitant lesions.

A mitral direct or pre-systolic murmur is present in a certain proportion of cases, either accompanied by a systolic mitral regurgitant murmur, or present without the latter. This murmur precedes and ends with the first sound; it has generally a peculiar, vibratory quality, and it is limited to a circumscribed space around the apex. Weakened aortic second sound and intensified pulmonic second sound are usually present, together with cardiac enlargement. These are the signs of contraction of the mitral orifice or mitral obstructive lesions.

The signs distinctive of mitral regurgitant and of mitral obstructive lesions are combined when these two varieties of mitral lesions coexist. Purring thrill is observed in a certain proportion of cases.

Symptoms and Pathological Effects.—Pain is rarely present. Abnormal force of the heart's action and palpitation denote consecutive enlargement, but these symptoms are often not prominent. The pulse is small and weak in proportion to the amount of either mitral obstruction or regurgitation, and in an advanced stage, it becomes irregular and intermitting; inequality of the pulse is, in some measure, characteristic of obstructive lesions. Turgescence of the jugular and other veins, lividity, or cyanosis, and dropsy occur at an advanced period when dilatation of the right cavities of the heart has been induced. Dyspnœa is more or less marked in proportion to the amount of regurgitation or obstruction, being more marked in cases of obstructive than regurgitant lesions. Cough and muco-serous expectoration occur frequently. Hæmoptysis is of frequent occurrence, and extravasation of blood in the lungs, or pulmonary apoplexy, takes place occasionally. Œdema of the lungs is a not infrequent event. All the symptoms and effects referable to the respiratory system are more marked when the lesions are obstructive than when they are only regurgitant.

In certain cases, lesions involving considerable and even great regurgitation or obstruction are remarkably latent and obscure as regards the symptoms and pathological effects. The diagnosis in these, as, in fact, in all cases, must rest mainly on the physical signs. On the other hand, lesions may exist, the existence and seat of which are determinable by physical signs, without involving much regurgitation or obstruction, and, consequently, not giving rise to any important symptoms or pathological effects. These lesions, so far as immediate danger is concerned, may be considered as innocuous.

DIAGNOSTIC CHARACTERS OF AORTIC LESIONS.

Physical Signs.—An endocardial systolic murmur is present in the majority of cases, with the traits which distinguish an aortic direct murmur; viz., its maximum of intensity at the base of the heart; the intensity diminishing as the stethoscope is carried downward over the body of the heart; comparatively feeble and often lost at the apex; propagated upward in the direction of the aorta, and into the carotids; not diffused over the left lateral surface of the chest; and if heard on the posterior surface, either limited to, or most intense in, the interscapular space on and above the level of the spinous ridge of the scapula. The murmur more or less intense; generally soft, but sometimes rough, and occasionally musical. This murmur is to be discriminated from an inorganic aortic murmur.

The aortic second sound of the heart is often weakened and indistinct; the pulmonic second sound is much less frequently intensified than in cases of mitral lesions. The mitral and tricuspid portions of the valvular element of the first sound retain their normal intensity, provided the lesions are limited to the aortic orifice. Enlargement of the heart exists in the majority of cases which come under observation. These are the signs of lesions with obstruction at the aortic orifice, that is, obstructive aortic lesions.

An aortic regurgitant murmur is present in a certain proportion of cases; it is often accompanied by a systolic aortic direct murmur, but it is not infrequently present without the latter;

its intensity is greatest near the left margin of the sternum, on or about the level of the fourth rib. The aortic second sound is impaired in proportion as the valves are injured. The pulmonic second sound is less frequently intensified than in cases of mitral lesions. Cardiac enlargement is usually present. These are the signs of insufficiency of the aortic valves, or aortic regurgitant lesions.

The signs distinctive of aortic obstructive and aortic regurgitant lesions are combined when these two varieties of aortic lesions coexist. Purring thrill is observed more infrequently than in cases of mitral lesions.

Symptoms and Pathological Effects.—Pain is oftener present than in cases of mitral lesions, but is often absent. Abnormal force of the heart's action and palpitation, as a rule, are more prominent symptoms than in cases of mitral lesions. The pulse, in cases of considerable obstruction, is not notably reduced in size and strength; it is rarely irregular or intermitting, and still more rarely unequal. In cases of aortic regurgitation it is quick, jerking, collapsing, and a longer interval than natural is sometimes observed between the apex-beat or systolic sound and the pulsation in remote arteries. Visible pulsation of superficial arteries is frequently marked. Turgescence of the jugular and other veins, and dropsy, occur at a later period than in cases of mitral lesions, and are much oftener wanting. Dyspnœa is less marked than in obstructive or regurgitant lesions of equal amount affecting the mitral valves and orifice. Cough and muco-serous expectoration and hæmoptysis are comparatively infrequent. Pulmonary apoplexy very rarely, if ever, occurs as a pathological effect. Œdema of the lungs is less frequent. All symptoms and effects, in fact, referable to the respiratory system, are less frequent and marked than in cases of mitral regurgitant, and still less than in mitral obstructive lesions.

Lesions affecting the aortic, as well as the mitral, valves or orifice, and involving considerable obstruction or regurgitation, are in certain cases remarkably latent and obscure as regards the symptoms and pathological effects. The diagnosis rests on the physical signs. Aortic lesions, also, may exist, and give rise to physical signs, without involving much or any obstruction or regurgitation, and are therefore innocuous as regards immediate danger.

The obstructive and regurgitant varieties of mitral and aortic lesions are found in various combinations in different cases. The diagnosis is then based on the union of the characters dintinctive of the varieties severally. The physical signs characteristic of each variety can generally be distinguished in these combinations.

DIAGNOSTIC CHARACTERS OF TRICUSPID LESIONS.

Physical Signs.—A systolic regurgitant murmur referable to the tricuspid orifice is rare even among the cases in which regurgitation through this orifice takes place. Regurgitation in consequence of widening of the tricuspid orifice, without a corresponding increase of the size of the valves, is not an uncommon result of enlargement of the right side of the heart consequent on mitral obstruction or regurgitation. The regurgitant current, however, rarely gives rise to a murmur, probably because the muscular power of the right ventricle, weakened by dilatation, is not sufficient to propel the current with force enough to produce an audible sound. For the same reason a murmur is not always present in the exceedingly few instances of tricuspid regurgitation occurring in consequence of organic changes analogous to those which affect the mitral valves. The rule, then, which is applicable to mitral lesions, viz., that a murmur is present in the vast majority of cases, cannot be applied to tricuspid lesions; and, hence, absence of murmur is not proof that the latter do not exist. A tricuspid regurgitant murmur, however, is sometimes observed. It is rarely intense or rough, and is usually low in pitch. Its maximum of intensity is at or above the xiphoid cartilage. It is heard within circumscribed limits, and is feeble or lost over the apex of the heart. Since tricuspid lesions, not congenital, are in most instances associated with lesions of one or more of the valves of the left side of the heart, a tricuspid regurgitant murmur, when present, accompanies a murmur, or murmurs, referable to the mitral or aortic orifice, or to both these orifices. It is to be distinguished from the latter by differences in pitch and quality, in addition to the difference of situation at which its maximum of intensity is observed.

Tricuspid regurgitation must diminish the intensity of the pulmonic second sound. If the tricuspid valves be injured, the tricuspid portion of the valvular element of the first sound must also be impaired.

A tricuspid direct murmur is one of the rare curiosities of medical experience. This murmur is best heard at or just above the xiphoid cartilage. I have never met with a case in which a tricuspid murmur existed without a corresponding mitral murmur. In the following case, there is reason to believe that these two murmurs were associated: A female patient was sent to my clinic by Dr. George K. Smith, of Brooklyn. She had an aortic direct, and a loud pre-systolic murmur. The patient remained under the observation of Dr. Smith, and died about a year afterward. On examination after death the aortic valves were found to be thickened and the free borders were studded with small vegetations. The mitral curtains were adherent at their sides leaving an opening which only admitted the end of the little finger. The tricuspid valves presented the same kind of lesions, the opening being contracted so as to admit only one finger. Both auricles were much dilated, the right more so than the left. The ventricles were moderately enlarged, dilatation predominating over hypertrophy.

Dr. Smith, who is an expert in auscultation, is convinced that, in this case, there was a tricuspid, as well as a mitral, direct murmur. His conviction is based on the fact that the pre-systolic murmur was heard, not only around the apex, but at, and to the right of, the ensiform cartilage.

Tricuspid obstructive lesions must diminish the intensity of the pulmonic second sound of the heart.

A systolic tricuspid regurgitant and a diastolic tricuspid direct murmur may be associated. I have, however, no practical knowledge of this combination.

Free regurgitation through the tricuspid orifice, with great dilatation of the right auricle and hypertrophy of the right ventricle, may occasion a strong impulse felt at the base of the heart, to the right of the sternum, simulating aneurism.

Symptoms and Pathological Effects.—Regurgitant and obstructive lesions, situated at the tricuspid orifice, do not produce those immediate effects on the respiratory system and the pulse which pertain to analogous lesions seated at the mitral orifice. They do not tend directly to give rise to dyspnœa, hæmoptysis, extravasation, &c., which are dependent on pulmonary congestion. They do not occasion irregularity, inequality, weakness, &c., of the pulse. Their immediate effects are manifested in the systemic venous system. Congestion of the systemic veins is a direct result proportionate to the degree of obstruction or regurgitation. Symptoms denoting this result are turgescence of the jugular and other veins; undulation and venous pulsation produced either by the contraction of the right ventricle, or by the auricular contraction, or by both; lividity due to accumulation in the venous radicles. A pathological effect of the congestion of the systemic veins is general dropsy. This effect occurs more directly and at a much earlier period when tricuspid lesions exist, than when it depends on dilatation of the right cavities consequent on valvular lesions situated at the left side of the heart. Cerebral apoplexy is more likely to be dependent on tricuspid than on mitral or aortic lesions, exclusive of the instances in which this affection proceeds from fibrinous plugs detached from within the heart-cavities.

DIAGNOSTIC CHARACTERS OF PULMONIC LESIONS.

Physical Signs.—Lesions situated at the pulmonic orifice may give rise to a murmur with the first sound of the heart, which, following the plan pursued in naming the mitral and aortic murmurs, should be called a pulmonic direct murmur. This murmur has its maximum of intensity in the second intercostal space on the left side of the sternum, the situation where the pulmonic second sound of the heart may be isolated from the aortic second sound. It may be propagated thence for a certain distance in a direction towards the left clavicle, but not in the direction of the aorta, and it is not heard over the carotids. The non-transmission into the carotids is a capital point in discriminating this murmur from an aortic direct murmur. The fact of the maximum of intensity in the second intercostal space is not distinctive, for this is sometimes true of an aortic direct mumur. To be considered as evidence of pulmonic lesions, not only must the murmur be referable to the pulmonic orifice, but it must be an organic murmur. Attention to the several points already considered, will enable the auscultator to determine that it is not inorganic. It has already been stated that pressure over the pulmonary artery in young subjects, with the stethoscope, will sometimes develop a murmur in this vessel. Pressure from some cause within the chest may also cause a murmur referable to this artery. It has been observed in cases in which the pressure on the vessel was produced by an aneurismal tumor, a morbid deposit within the pericardium, enlarged bronchial glands, and a solidified portion of lung.[1] It must be difficult, in some instances, to eliminate these several sources of fallacy. A pulmonic murmur may be quite intense. I have met with an example of a musical murmur, systolic and diastolic, persisting through the whole beat, in other words, continuous, distinctly referable to the pulmonic artery, and so loud as to be heard with the ear in close proximity to, without being in contact with, the walls of the chest.

A diastolic murmur may accompany insufficiency of the pulmonic valve, constituting a pulmonic regurgitant murmur. It must be difficult to distinguish between this and an aortic regurgitant murmur, except the murmur be accompanied by a pulmonic direct murmur, and not by an aortic direct murmur. In the vast majority of the cases in which a diastolic murmur is present, it is either an aortic regurgitant, or a mitral direct murmur.

Lesions involving injury to the pulmonic valve must impair the intensity and distinctness of the pulmonic second sound of the heart.

A pulmonic direct and a pulmonic regurgitant murmur may be associated in the same case, or either may be present without the other. Pulmonic lesions, however, exclusive of congenital malformations, are so rare, that the opportunities of any clinical observer, however large his experience, for studying the physical signs, are extremely limited. Hypertrophy of the right ventricle, which is produced by obstructive or regurgitant lesions of the pulmonic orifice, involves augmented intensity of the tricuspid valvular element of the first sound, and an impulse in the epigastrium.

A remarkable case in which a continuous loud murmur was produced at the pulmonic orifice, occurred in the practice of Dr. William M. Chamberlain, of New York. The patient, a broker's agent, aged about 40, had long suffered from palpitation, but continued to pursue his business which required considerable activity. A fortnight before his death he became suddenly ill with chills, increased disturbance of heart, dyspnœa, and general malaise. I saw him with Dr. Chamberlain a few hours before his death. The physical signs were as follows: The apex-beat was in the sixth intercostal space, without the linea mammalis, and the superficial cardiac space was increased. Cardiac impulses were felt in all the intercostal spaces between the base and apex. A well-marked purring thrill was felt when the hand was placed over the præcordia. At the apex there was no murmur. At the base, on the right side of the sternum, there was no murmur. There was no murmur in the carotids. Between the third and fifth rib, on the left side of the sternum, there was an extremely loud, rough, continuous murmur. Mitral and aortic lesions being excluded by the foregoing results of physical exploration, the existence of some anomalous condition was inferred, and aneurism of the heart was suspected.

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[1] Bellingham, *op. cit.*, part ii, p. 386. Da Costa in Am. Journ. of Med. Sciences, January, 1859.

The autopsy revealed a cavity of the size of a pullet's egg between the pulmonary artery and the aorta, with an opening of the size of a crow's-quill into the right ventricle just below the sigmoid valves. The opening was evidently of recent date. The cavity was empty, and lined by a membrane which was evidently of old date. No other opening into this cavity existed. The pericardium was universally adherent by firm adhesions. The heart was considerably enlarged, the enlargement being chiefly seated in the left ventricle, and dilatation predominating. The aortic, the pulmonic, the tricuspid, and the mitral valves were healthy. The left ventricle was filled with soft, black clots. The right ventricle contained a white clot closely adherent to the tendinous cords and the trabeculæ.

The passage of the blood into this cavity during the ventricular systole, and its return into the ventricle during the diastole, plainly occasioned the loud continuous murmur. It was concluded that, prior to the production of the cavity, there was a collection of liquid, derived from the pericardial sac, which accumulated in this situation in consequence of the pericardial adhesions; and that, perforation taking place, the liquid was evacuated into the right ventricle. This collection of liquid must have formed a tumor pressing upon the aorta and pulmonary artery; hence, the enlargement of the heart. The perforation and discharge of the liquid into the ventricular cavity probably took place at the time of the sudden illness a fortnight before the death of the patient.

The right pleural cavity contained a large quantity of dropsical effusion, and there was also considerable effusion into the left pleural cavity. The liver was much enlarged. There were not multiple abscesses in this organ, or elsewhere, denoting pyæmia.

Symptoms and Pathological Effects.—The primary effect of obstructive or regurgitant lesions situated at the pulmonic orifice is enlargement of the right ventricle. The secondary and remote effects, and the symptoms thereon dependent, are essentially those which are occasioned by tricuspid lesions, being due to distension of the right auricle, tricuspid regurgitation, and congestion of the systemic veins.

TREATMENT OF LESIONS AFFECTING THE VALVES AND ORIFICES OF THE HEART.

With reference to the management of patients affected with chronic valvular lesions, several important considerations, which have been already presented, are to be kept in mind.

1. The anatomical changes which the valves and orifices have undergone are irremediable, and therefore do not claim any special medicinal treatment. The existing lesions must remain. The damage which they have occasioned cannot be repaired. Medication employed for that object will be worse than useless. The morbid processes giving rise to the lesions have occurred long before the symptoms of an organic affection of the heart became developed. In the majority of cases the origin of the affection may be dated at an attack of acute rheumatism several years prior to the period when ailments referable to the heart are first experienced. The changes incidental to these processes have, in the meantime, been slowly progressive. They will, probably, continue to progress, involving more and more damage. This we cannot expect to prevent, but something can be done to retard their progress, and more especially, to control their primary effects.

These facts not being always sufficiently appreciated, practitioners sometimes employ mercury and other remedies called alteratives, with a view to the removal of morbid material deposited on or about the valves. I have met with cases in which depletion, low diet, counter-irritation, &c., were resorted to, under the idea that the lesions involved persisting chronic inflammation of the endocardial membrane. These measures cannot fail to aggravate the cardiac symptoms, and to expedite effects which it is a great object of the management to postpone as long as possible.

2. Lesions may exist, giving rise to murmurs more or less intense, but without producing any immediate morbid effects, inasmuch as they involve little, if any, obstruction or regurgitation, and consequently have not led to enlargement of the heart. Such lesions may remain for an indefinite time latent or innocuous, but there is a probability of their leading, at some future period, to serious results. These cases, therefore, claim a certain amount of watchfulness and supervision. The existence of a cardiac murmur dependent on latent or innocuous lesions is often ascertained by accident, there being no symptoms of disease referable to the heart. I have repeatedly met with it in examining persons who considered themselves in perfect health. These persons are in no immediate danger, and it would give rise to needless alarm to inform them that they have an organic affection of the heart, since it is a common notion that any such affection involves liability to sudden death. There is, however, a prospective danger not to be overlooked. In examinations for life assurance it is not uncommon for organic murmurs to be discovered when there had been no ground for the suspicion of any cardiac disease. In such cases, although the heart be not enlarged, and there are no present symptoms of disease, the application for assurance should undoubtedly be rejected in view of the prospective danger incident to enlargement.

3. Even when lesions exist which, involving more or less obstruction or regurgitation, have induced considerable enlargement of the heart, it by no means follows that the immediate danger is great. I have known many persons who for many years have had valvular lesions with more or less enlargement of the heart, but who did not consider themselves as invalids, and some of whom were engaged in active business.[1] In such cases

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[1] *Vide* Essay on Clininal Study of Heart-Sounds, Trans. Am. Med. Association, vol. xi.

the organic affection does not call for active therapeutical measures; but knowledge of the existence of the cardiac affection is highly important to the practitioner, and should influence his advice as regards habits, regimen, &c., as well as his treatment of intercurrent diseases. The tolerance of lesions in some instances is truly remarkable. For example, a boy, aged eleven years, presenting three organic murmurs, viz., a mitral regurgitant, an aortic direct, and an aortic regurgitant, with much cardiac enlargement, the præcordia projecting and the apex beating half an inch without the nipple, not only made no complaint of symptoms referable to the heart, but was able to take violent exercise, and to engage in rough sports with apparently as much ease as any of his companions. My records furnish many instances of a similar character. In these cases the physical signs denote, in association with the valvular lesions, enlargement by hypertrophy, not by dilatation, and the action of the heart is vigorous.

4. The enlargement of the heart being dependent upon, and, as a rule, proportionate to, the amount of obstruction or regurgitation, or both, occasioned by valvular lesions, the abnormal size of the heart may, in general, be taken as a criterion of the importance to be attached to the lesions. As long as valvular lesions exist without having induced enlargement of the heart, they give rise, as a rule, to no appreciable evil consequences, if the liability to embolism, from plugs of fibrin or detached morbid growths from within the heart, be excepted. The primary morbid effects of these lesions are exerted upon the heart, ending sooner or later, in its enlargement; and the symptoms of cardiac disease represent secondary and remote effects of the valvular lesions, the latter effects following, and in a great measure depending upon the enlargement induced by the lesions.

5. The secondary and remote effects of valvular lesions, as a rule, are not developed so long as the enlargement of the heart is by hypertrophy, unless, from fatty degeneration or some other cause, weakness of the organ has been induced. Obstructive and regurgitant lesions tend first, as a rule, to produce hypertrophy. The muscular walls increase in thickness up to a certain limit. When this limit is reached, dilatation of the cavities ensues, and, finally, predominates over the hypertrophy. The increased power of the organ, incident to the hypertrophy, com-

pensates for the immediate consequences of obstruction and regurgitation. The hypertrophy is thus a conservative provision to obviate the evils of obstructive and regurgitant lesions. The patient is comparatively safe as long as hypertrophy predominates. The secondary and remote effects are incident to the dilatation which takes place after the hypertrophy has reached its limit. The immediate danger, other things being equal, is proportionate to the amount of predominance of the dilatation. This is because the heart is weakened in proportion to the predominance of dilatation. Weakness of the organ, due to fatty degeneration or other causes than dilatation, will also favor the development of the secondary and remote effects of valvular lesions. These facts are of great importance in their bearing on the treatment of patients affected with these lesions.

In view of the foregoing considerations, the main objects of treatment which relate directly to the condition of the heart, in the early stage of valvular lesions, are, 1st. To prevent, or, as far as possible, to retard, the progressive anatomical changes incident to the existing lesions; and, 2d. To obviate the tendency to weakness and dilatation of the heart.

The anatomical changes seated in the valves and orifices give rise to the varied morbid appearances which have been mentioned[1] in Chapter III. These changes cannot be reached by any special remedies. Their progress can only be indirectly affected by preventing overstraining of the valves, which must occur whenever the organ is unduly excited or overtasked, and by avoiding the causes which favor renewal of inflammation of the endocardium. Excessive muscular exercise, great mental excitement, the intemperate use of alcoholic stimulants, &c., promote the progress of valvular lesions, by exciting unduly and overtasking the heart. Patients with valvular lesions devoid of immediate danger, should pursue a course of life which, as far as practicable, will be exempt from causes inducing great disturbance of the circulation. In pointing out the regimen, habits, &c., however, the importance of fostering the muscular power of the heart, to which reference will be presently made, is not to be lost sight of. The causes favoring the development of endocarditis, are, in general, those which tend to give rise to rheumatism, since in the vast majority of cases endocardial inflammation is of rheumatic origin. Unusual exposure to the vicissitudes of the weather are regarded, perhaps justly, as often determining an attack of rheumatism when the diathesis exists. The existence of the diathesis is shown by the previous occurrence of one or more rheumatic attacks in the majority of the persons affected with valvular lesions, the origin of the latter being referable, in such cases, to a former attack of rheumatism. A fresh attack, exposing the patient to a renewal of the endocarditis, is a calamity to be averted, if possible, by avoiding the exciting causes as far as these are appreciable. In fulfilling this object of treatment, hygienic regulations are chiefly involved. Judicious management will undoubtedly do something toward rendering the progress of the lesions more slow than would otherwise be the case; but we can hardly expect to arrest their progress. If, however, they are very slowly progressive, life and comfortable health may be prolonged for an indefinite period, perhaps even to an advanced stage.

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[1] *Vide* page 128.

The same hygienic regulations are equally important with reference to the second object, viz., to obviate the tendency to weakness and dilatation of the heart. The judicious management of patients affected with valvular lesions prior to the development of the secondary and remote effects of these lesions, depends, in a great measure, on a proper appreciation of this object. It has been said that the treatment of valvular lesions virtually resolves itself into that designed to prevent and diminish enlargement of the heart. This involves an important error as well as an important truth. It is highly desirable to prevent dilatation but not to arrest hypertrophy. On the contrary, if enlargement must occur as a result of obstructive or regurgitant lesions, hypertrophy is to be encouraged, if by so doing, dilatation may be prevented. The serious evils of valvular lesions, as we have seen, occur when the limit of hypertrophy has been reached and dilatation predominates. In the predominance of hypertrophy may be said to consist, in a great measure, the security of the patient. This remark is also applicable to the muscular power of the heart. So long as the organ acts with vigor, the secondary and remote evils are deferred. Weakness of the heart leads to these evils. Weakness predisposes to dilatation, and, conversely, dilatation involves weakness. To prevent weakness and dilatation, then, in the early stage of valvular lesions, is the great object of treatment so far as it relates directly to the condition of the heart.

Undue excitement and overtasking of the heart induce weakness and favor dilatation. The muscular power here, as in other situations, is exhausted by too great exertion, and the walls yield more readily to distension, under these circumstances, from the accumulation of blood within the cavities. The causes, already referred to, which excite unduly and overtask the heart, viz., excessive muscular exercise, mental excitement, the intemperate use of alcoholic stimulants, &c., are, therefore, to be avoided with respect to the second, not less than the first, object of treatment. Exercise, however, within certain limits, is highly important with a view to the preservation of the power of the heart's action. Patients affected with obstructive or regurgitant lesions will retain a compensatory vigor of the heart, and the epoch when dilatation succeeds hypertrophy will be postponed, for a longer period by habits which involve a judicious amount of exercise than by a life of complete repose. Active occupations, whether pursued as a calling or for amusement, or with reference merely to exercise, should not be abandoned. Persons under the necessity of performing daily manual labor will do better to continue to work, as far as they are able without inconvenience, than to become fixtures in the wards of a hospital. They who are above this necessity should either follow some active pursuits or engage in sports which demand a certain amount of physical activity. Indolence or inaction of the muscular system tends to produce weakness of the heart and favors fatty degeneration, thereby contributing to the production of dilatation rather than hypertrophy. The rules which should govern exercise have already been considered in connection with the treatment of hypertrophy, to which the reader is referred.[1] These rules are applicable to cases of valvular lesions, with or without hypertrophy. They are, of course, not to the same extent applicable to cases in which the lesions have already led to dilatation.

The diet suited to obviate a tendency to weakness and dilatation is that best adapted to healthy nutrition. Healthy nutrition, and thereby the muscular vigor of the heart, require blood rich in nutritive materials. A poor and insufficient diet tends to hasten the evils resulting from valvular lesions. The diet should embrace a fair proportion of animal food. Liquids should be taken sparingly, the object being to secure a good quality, but not to increase the quantity, of blood. Restrictions, as respects fatty substances and those readily converted into fat, are important if there be grounds to suspect a disposition to fatty degeneration. The articles of food should be adapted to the digestive powers. The action of the heart, as is well known, is liable to be disturbed through its sympathetic connection with the stomach, when digestion is labored or imperfect. Dyspeptic disorders will claim appropriate treatment. Tonics and stimulants, in moderate quantity, are indicated whenever the digestive powers are enfeebled. Exercise in the open air, within proper limits, is important with reference to its influence on digestion. Cheerfulness and mental recreation are desirable for the same end.

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[1] Chap. I, page 77.

Opposite conditions of the blood alike tend to weakness and dilatation, viz., plethora and anæmia. If the blood be too abundant and the red globules in excess, the heart is overtasked and unduly stimulated. Bloodletting, under these circumstances, may sometimes be appropriate. But it should be employed with discrimination and great circumspection, inasmuch as the impoverishment caused by its injudicious employment is a condition worse than plethora. In general, other methods of depletion, which are not spoliative, are to be preferred, viz., saline laxatives and diuretics, in conjunction with a dry diet. Anæmia is a far more unfavorable condition than plethora, and claims efficient treatment with chalybeate tonics, nutritious diet, &c. The symptoms referable to the heart, in some cases of valvular lesions, are, in a great measure, due to functional disorder incident to anæmia, and when the anæmic condition is removed, all the symptoms may disappear. This fact should be borne in mind. The practitioner is liable to consider all the symptoms as resulting directly and exclusively from the lesions, and, consequently, is led to exaggerate the immediate danger from the latter. Patients who suffer much from palpitation, &c., when anæmia is conjoined with valvular lesions, may experience no inconvenience when the blood is restored to its normal condition. This is intelligible in view of the well-known fact that anæmia often gives rise to functional disorder of the heart when this organ is free from organic disease.

The treatment of valvular lesions, as thus far considered, has reference to the condition of the heart prior to the period when dilatation has ensued, either enlargement of the organ not having taken place, or hypertrophy being as yet predominant. The secondary and remote effects of valvular lesions, as has been seen, for the most part occur after dilatation predominates over hypertrophy. It remains to notice the treatment due to the condition of the heart at this stage, and the treatment of the secondary and remote effects.

So far as the heart is concerned, the treatment at this stage is essentially that which has been already considered in connection with the subject of dilatation.[1] Extrinsic circumstances affecting the circulation, such as exercise, mental emotions, &c., now occasion symptoms of disturbance much more marked, and attended with far greater inconvenience. The ability to take exercise without palpitation and dyspnœa is diminished, and quietude may be indispensable. Within the limits, however, to which exercise may be borne without discomfort, it is still desirable. A nutritious, sustaining diet is not less indicated. Attention to the condition of the stomach is equally important. Bloodletting is much more rarely, if indeed it be ever, called for. Plethora, if it exist, claims methods of depletion which are not spoliative. Anæmia demands the same efficient measures. The general object is to increase, if possible, the muscular power of the heart. It is doubtful whether this object is promoted by any remedies which exert a direct, special effect upon the heart. Nux vomica or strychnia and arsenic, given in minute doses and long continued, have been advocated as remedies having such an effect.

At this stage, not only is the heart enfeebled, but the rhythm of its action is often disturbed, as denoted by irregularity, intermittency, and inequality of the pulse. Remedies designed to tranquillize and regulate the movements of the organ are now indicated. For this end, digitalis often proves a valuable remedy. Care is to be taken not to give this remedy to the extent of retarding too much the heart's action. Observing proper caution in this respect, the action of the heart not only becomes more regular under its use, but the contractions of the ventricles appear to take place with greater power and completeness, as denoted by increased fulness and force of the pulse. It is proper to add that this statement, as regards the value of digitalis, is in opposition to the views of some authors who regard it as rarely useful and attended with hazard. Its usefulness and freedom from danger turn on the influence which it exerts on the power of the heart's action. The opinion which I have expressed is based on inferences drawn from clinical observations. Belladonna, the hydrocyanic acid, aconite, and sometimes opium in small doses, are other remedies which may be found useful in fulfilling this indication.

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[1] *Vide* Chap. I, page 87.

A large share of the secondary and remote effects of valvular lesions are dependent on passive congestion. The lungs are generally first and most affected; afterward, the brain and abdominal viscera. The tendency to congestion of internal organs is obviated most effectively by measures which prevent weakness and dilatation of the heart, or which increase its muscular power if these primary effects have already taken place. In addition, something may be effected by promoting, as far as possible, the circulation in the extremities and at the surface of the body, and by revulsive measures. The body should be protected by sufficiently warm clothing, and prolonged exposure to cold should be avoided. Friction of the surface and stimulating pediluvia are useful in fulfilling this indication. Rubefacient applications and dry cupping are the appropriate revulsives.

Dyspnœa, cough, and expectoration often claim special attention. The suffering from a sense of breathlessness, frequently severe, must be palliated by the remedies known as antispasmodics, particularly the ethereal preparations, and by anodynes, in addition to revulsive measures. Palliation, in most instances, is all that can be expected from treatment. Cough, exceeding that required for expectoration, may be allayed by such remedies as belladonna, hyoscyamus, conium, hydrocyanic acid, or by small doses of opium. Muco-serous expectoration is sometimes a mode of relief, being a sort of local, spontaneous depletion, and is to be encouraged rather than arrested. Superinduced or intercurrent pulmonary affections, such as bronchitis, pneumonia, and pleurisy, demand appropriate treatment, but bloodletting and other measures which tend to weaken the heart, are to be employed with great circumspection. The coexistence of valvular lesions with dilatation generally renders sustaining measures more than ever important in the management of these affections.

The importance of correcting disorders of digestion and improving this function when impaired, is not less in the advanced than in the early stage of valvular lesions. Mercury is often prescribed with a view to relieving congestion of the liver by increasing the secretion of bile. Granting that it may have this effect, it is a remedy of more than doubtful propriety if given so as to incur risk of mercurialization. As an occasional laxative or cathartic it is admissible. Constipation is to be avoided, and moderate purgation, from time to time, affords relief as a means of local depletion when the abdominal organs are suffering from congestion. Purgatives too often repeated, however, will do harm by depressing the vital powers, and thereby weakening the heart.

For the relief of cerebral congestion, which we have seen occasions inconvenience and evils less frequently than is generally supposed, reliance must be had on the revulsive measures, in addition to those which relate directly to the condition of the heart.

General dropsy is a remote effect occurring in a large proportion of the cases of valvular lesions which are prolonged to an advanced stage. It is usually evidence of a degree of weakness and dilatation precluding expectation of permanent improvement, and denoting that a fatal termination is not far distant. But in some cases complete relief is obtained, and the dropsy may not recur for a considerable length of time. These are cases in which the dropsy has been promoted by associated morbid conditions, such as impoverished blood, or by extrinsic causes which have temporarily enfeebled the heart. The event is not only important as a symptom, but it imposes inconvenience and suffering proper to itself, and hastens a fatal issue. It calls, therefore, for appropriate treatment.

The immediate objects of the treatment of cardiac dropsy are the resorption of the effused liquid, and, at the same time, increased power and completeness of the ventricular contractions. Resorption is to be effected, if possible, by eliminating water from the blood by means of diuretics or hydragogue cathartics, conjoined with a dry diet. In the selection of diuretics, those are to be preferred which increase the quantity of urine without increasing, proportionably, its solid constituents; in other words, those which eliminate especially water. Experimental observations render it probable that different diuretics differ in this respect, digitalis, juniper, and squill, for example, increasing the flow of urine, while the amount of solid matter is below that of health.[1] In the employment of diuretics and hydragogue cathartics, more especially the latter, great care is required not to push the remedies to an extent to lower too much the powers of the system, and thereby weaken the action of the heart. While measures are pursued to effect resorption, the second object of treatment should not be lost sight of, viz., to increase the power and completeness of the ventricular contractions. This object involves nutritious diet, tonics, and exercise within proper limits.

The choice between diuretics and hydragogue cathartics will depend on the readiness and extent to which the kidneys respond to the former of these two classes of remedies. Different cases differ much in this respect. In some instances hypersecretion of urine is easily effected; in other instances with difficulty, and not to the extent desired. Reliance must then be had on cathartics. In general, diuretics should be first tried, and they are to be preferred if found to operate satisfactorily. The general principle involved in the selection of diuretics has been stated, but we have not, as yet, sufficient facts to establish a division of all the numerous articles which induce diuresis into those which do and those which do not increase the solid constituents of the urine. As regards the diuretic effect, we have to be guided, in a great measure, by experimental trials in individual cases. A diuretic remedy may act efficiently in one case and prove inefficient in another case, a different article being found to act satisfactorily in the latter. All practical physicians must have been led to notice this fact. Usually, different diuretics act better in combination than separately. Digitalis and squill, for example, may be given in combination, and, at the same time, the bitartrate, the nitrate, or the acetate of potash, largely diluted in an infusion of juniper, parsley, or fleabane. As remarked by Prof. Wood, diuretics may fail at a particular period, and act efficiently at another period in the same case. The ingestion of liquids should be restricted so far as due regard to comfort will permit. The mode by which diuretics lead to resorption being the elimination of water from the blood, which involves an increase of the density of the latter, it is plain that their efficacy in relieving dropsy will be limited or rendered nugatory by the free introduction of liquid into the system.

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[1] On the Action of Certain Vegetable Diuretics. By William A. Hammond, M.D., Am. Journ. of Med. Sciences, Jan., 1859.

Prof. Christison has advocated the external use of diuretics in certain cases. I have repeatedly tried this method, and generally without much success. In one instance, however, which came under my observation, the patient being under the care of my friend, Prof. J. P. White, the effect was remarkable. Diuretics given internally having lost their effect in this case, a liniment composed of equal parts of the tinctures of squill, digitalis, and iodine, and two parts of soap liniment, was applied freely, with considerable friction, over the abdomen and thighs. The patient took no remedy in addition except the iodide of iron. Under this treatment, in a week, he lost nine pounds in weight, the secretion of urine being greatly increased. The anasarca disappeared, and did not again return for several months, the comfort and general health of the patient becoming in the meantime much improved.

Hydragogue cathartics are to be employed when diuretics fail to act satisfactorily or to accomplish the end desired. Of the different articles embraced in this variety of cathartic remedies, elaterium is by far the most efficient. The great activity of this article, when pure, requires care in its administration. From a sixth to a quarter of a grain may be directed every one or two hours till a sufficient number of watery stools are procured. The intervals between the days of its administration must be regulated by the state of the patient and the prostration occasioned by its operation. The bitartrate of potash, given in pretty large doses in a concentrated solution, frequently acts efficiently. This remedy with jalap forms an efficient combination.

The treatment of cardiac dropsy is sometimes remarkably successful. The effused liquid is rapidly and completely absorbed. The patient experiences so much relief that he is encouraged to hope for recovery. This the physician does not expect, but he may hope to postpone the recurrence of dropsy by strengthening the heart and removing causes which exist in addition to the obstruction due to the cardiac affection. Anæmia predisposes powerfully to dropsical effusion. The restoration of the blood to its normal condition may secure long exemption from recurrence of the dropsy. If, however, the dropsy be altogether dependent on the obstruction caused by the cardiac affection, some degree of palliation is all that is to be hoped for. The accumulation of effused liquid remains and augments, often, notwithstanding appropriate measures of treatment. This is more likely to be the case if the patient be obliged to remain much of the time in the sitting posture on account of dyspnœa. The coexistence of Bright's diseases also lessens greatly the prospect of relief. Temporary relief is sometimes obtained by puncturing the lower extremities, water draining away in abundance through the artificial openings. Numerous minute punctures may be made with a needle or pin, not deep or large enough to occasion either pain or hemorrhage. These may be frequently repeated. I have not observed unpleasant results from this mode of making punctures. If the size of the punctures be sufficient to cause visible wounds, there is a liability to erysipelatous inflammation and gangrene, which renders the operation of doubtful expediency. This remark is also applicable to incisions, which some writers have advised. The great distension of the integument of the lower extremities in some instances gives rise to fissures and ulcerations through which the effused liquid freely escapes. When these occur, it is not wise to attempt to heal them promptly.

An important part of the management of cases of valvular lesions relates to the communications on the subject to be made to patients. An endocardial organic murmur which is discovered incidentally in the examination of a patient, need not be announced, for, if the heart be not enlarged, the danger is prospective, perhaps remote, and gratuitous uneasiness may be occasioned by the patient being made acquainted with the existence of an organic affection. Unsoundness of the heart is generally supposed to be, in all cases, a very serious matter, and to involve liability, at any moment, to sudden death. Some practitioners, participating in this popular impression, injudiciously apprise patients that they must expect to be taken off without warning. I have met repeatedly with instances in which persons have been so informed, much to the prejudice of their comfort, usefulness, and even their prospect of preserving comfortable health for a long period. It should be borne in mind that lesions which give rise to murmurs are often innocuous, the danger being prospective, and perhaps remote. And even when the lesions are of a nature to involve obstruction or regurgitation, and have led to considerable enlargement of the heart, life and comfortable health may be preserved for many years. Moreover, statistics show that sudden death occurs in only a small proportion of the cases of organic disease of the heart, sometimes involving, when it does occur, some associated morbid condition—for instance, structural degeneration of the cerebral arteries, leading to rupture and extravasation. In cases of apoplexy proving suddenly fatal in persons with cardiac lesions, the sudden death, if the head be not examined post mortem, is apt to be imputed wholly to the condition of the heart.

If the attention of the physician be called by the patient to the state of the heart, and an opinion requested, the existence of lesions cannot be denied. Truth, and justice to the physician himself, as well as good faith toward the patient, require that the fact should be candidly stated. The statement then should be accompanied by such explanations as will serve to divest the fact of greater importance than really belongs to it. If proper pains be taken, this, happily, is not difficult, since the mental condition incident to disease of the heart generally leads patients to accept the most favorable view of the case which can be conscientiously submitted.

The prognosis, to the friends of patients, should be cautiously given. The duration of life, except in cases of advanced disease, is extremely variable. There is, on the one hand, a liability to certain accidents and to incidental affections which may prove fatal unexpectedly; and, on the other hand, patients often live for a long time after the signs and symptoms denote lesions of a most serious character.

As regards a liability to sudden death, the prognosis is of much importance in the communications of physicians to the friends of patients, and sometimes to the patients themselves. Sudden death is attributable directly to disease of the heart in a certain proportion of cases, the proportion being small, as just stated. There is a wide difference between aortic and mitral lesions with respect to a liability to sudden death. Aortic lesions exist in a large majority of the cases in which sudden death is attributable directly to disease of the heart. Of a collection of cases analyzed by Aran, the number in which aortic lesions existed alone, exceeded by more than four times the number in which the lesions were mitral, the proportion being as six to twenty-five. Of 16 fatal cases, in my collection, in which aortic lesions existed alone, sudden death, attributable directly to the heart, occurred in 6; whereas, of 15 fatal cases in which the lesions were exclusively mitral, sudden death occurred in only 2.

Of 14 fatal cases in which both mitral and aortic lesions existed, sudden death did not occur in a single case. It is, therefore, especially in cases of aortic, not associated with mitral, lesions that there is a liability to sudden death.

Aortic lesions rarely occasion sudden death before they have induced considerable dilatation of the left ventricle. When dilatation has taken place the rationale is intelligible. The left ventricle, weakened by the dilatation, is habitually unable to expel with the systole the blood which it contains. Now, if from any cause the accumulation of blood within it is suddenly much increased, the walls are further weakened by this increased accumulation, and if it go on beyond a certain limit they become paralyzed, the systole is arrested by the distension, and instantaneous death occurs. It is easy to understand that a few seconds may suffice for this result, in view of the rapid accumulation of blood in the ventricular cavity from a mitral direct and an aortic regurgitant current combined. Aortic lesions which involve notable insufficiency of the semilunar valves, are especially those which render the patient liable to sudden death. This liability, it is obvious, is much increased by the coexistence of fatty degeneration of the left ventricle, the heart being weakened by this change added to the dilatation. Coexisting mitral lesions are conservative, as regards sudden death, because the obstruction or regurgitation, or both, which they involve, prevents the over-distension and consequent paralysis of the left ventricle.

The foregoing explanation of sudden death from aortic lesions seems adequate without resorting to the ingenious hypothesis of a French writer, Mauriac. This writer assumes that the blood is not forced into the coronary arteries during the ventricular systole, partly because the semilunar valves, pressed against the walls of the aorta, obstruct the orifices of these vessels, and, in part, because the arteries in the muscular walls are compressed by the contraction of the muscular fibres during the systole; but during the diastole, the semilunar valves arresting the backward current, the blood is driven into the coronary arteries by the force of the recoil of the coats of the aorta, the arteries in the muscular walls being then free from pressure owing to the passive condition of the muscular fibres. Now, in certain cases of aortic lesions, the obstruction at the orifices of the coronary arteries is increased by calcareous matter, and if the semilunar valves be notably insufficient, the force of the recoil of the coats of the aorta fails to drive the blood into the coronary arteries owing to the free regurgitation into the ventricle. Under these circumstances, the ventricle is weakened by an insufficient supply of arterial blood. Moreover, the walls of the ventricle contracting feebly and imperfectly, the blood within the veins of the ventricle is imperfectly expelled, and hence, another cause of weakness of the ventricular walls is venous congestion. These causes, perhaps, in some cases, co-operate in determining the occurrence of sudden death by paralysis from over-distension of the left ventricle.[1]

Sudden death in some cases in which the lesions are mitral is attributable to paralysis from over-distension of the right ventricle. It is easy to understand that there may be a sudden increase in the accumulation of blood in the right ventricle, and that the systole of this ventricle weakened by dilatation, and perhaps, also, by fatty degeneration, may be arrested in the same way as the left ventricle under similar circumstances.

The coagulation of blood within the cavities of the heart is an occasional cause of a rapidly fatal termination; but death from this cause is not sudden, limiting the application of this term to cases in which patients die instantly, or after a few moments, as when rupture of the heart takes place, or an aneurism bursts into the pericardial sac. Anæmia of the medulla oblongata has been supposed to be a condition giving rise to sudden death in some cases.

In conclusion, it is to be borne in mind that the liability to sudden death is incident especially to aortic lesions, when these are not associated with mitral lesions, and when there is, in addition, notable weakness of the heart from dilatation or fatty degeneration. Under these circumstances, in addition to measures of treatment with a view to regulating and increasing the power of the heart's action, it is important that patients should, as far as possible, avoid muscular exertions, moral emotions, and other causes of a sudden and considerable increase of the accumulation of blood within the cavities of the heart.

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[1] Nouveau Dictionnaire, Paris, 1868, Art. Cœur.

CHAPTER V.

CONGENITAL MISPLACEMENTS, DEFECTS, AND MALFORMATIONS OF THE HEART.

Transpositions and expositions—Ectopia pectoralis cordis—Ectopia cordis ventralis—Ectopia cordis cephalica—Deficiency of the pericardium—Biloculate heart—Heart with three cavities—Deficiency of auricular and ventricular septa—Obliteration of, and obstruction at, the pulmonic artery—Supernumerary septum—Patency of the foramen ovale and the ductus arteriosus—Deficiency and excess of segments of semilunar valves—Union of curtains of the mitral and tricuspid valves—Diagnosis of malformations—Causes of death—Coexistence of tuberculosis—Treatment.

CYANOSIS. Definition—Its connection with different malformations—Conclusions respecting the mode of its production—Cyanotic phenomena in various affections exclusive of malformation of the heart—Diversities as respects degree and extent of the cyanotic discoloration—Other variations—Associated symptoms—Diagnosis—Prognosis—Treatment.

THE various malformations, &c., of the heart are especially interesting in their relations to the physiology of the circulation and to embryology. A general knowledge of this subject, however, is important to the physician. It is desirable for the practitioner to recognize the existence of congenital affections, by means of signs and symptoms, during life, and, as far as practicable, to discriminate between them. The study of certain of these affections has an important bearing on a subject which will, in consequence, be considered in this chapter, viz., cyanosis. Treating of the different varieties of congenital affections only so far as is consistent with the practical scope of this work, they will occupy but a small space.

CONGENITAL MISPLACEMENTS OF THE HEART.

The misplacements are either of *transposition* or *exposition*. Instances in which the heart is situated in the right side of the chest, are not so infrequent but that numerous examples have been reported. In some instances the other viscera are likewise transposed, and sometimes the situation of the heart alone is abnormal. This abnormity is not incompatible with health and long life. Removal of the heart into the right side, as is well known, occurs as a result of pleurisy, with a large amount of effusion, affecting the left side. The organ may form attachments and remain fixed in the right side after the liquid effusion has been absorbed. A similar result sometimes follows absorption of a large amount of liquid effusion into the right pleural sac. I have met also, with several cases in which, as a consequence of great destruction of the right lung from tuberculous disease, the heart had been drawn by suction into the right side. The existence of pleuritic effusion is readily ascertained by means of the physical signs, and the permanent changes in the size and configuration of the affected side enable the diagnostician to determine the fact of its previous existence. When the displacement is due to tuberculous disease, the evidence of the latter is sufficiently obvious. These cases of misplacement of the heart are to be discriminated from those in which it is congenital.

In cases of *exposition*, the heart may be situated exterior to the chest. These instances constitute the variety known as *ectopia pectoralis cordis*. In none of the instances cited by Peacock in which the heart alone was exterior to the chest, did life continue over forty hours after birth. This variety of ectopia is extremely rare, excluding the cases in which, at the same time, the viscera of the abdomen are protruded. They offer valuable opportunities for studying the movements of the heart by means of the sight and touch. The heart, however, may be covered only by the integument, owing to a deficiency of the walls of the chest in this situation, and in these cases life may continue indefinitely. In a case reported by Ramirez, a Mexican physician, the patient had attained to the age of seventy-five years. In another variety of exposition, the heart is situated below the diaphragm, and in these cases there may, or may not, be an external tumor. This variety, called *ectopia cordis ventralis*, is not incompatible with long life and vigorous health. In another curious variety, the heart is situated in the front of the neck. This is called *ectopia cordis cephalica*. In the cases reported of this variety, the infants have died shortly after birth. These different misplacements may be readily ascertained by inspection, palpation, and auscultation. But in cases of *ectopia cordis ventralis*, the malposition may occasion so little inconvenience that attention may not have been directed to it during life, and the anomaly, therefore, is not ascertained before death. A French physician, Deschamps, has reported the case of an old soldier, in which, on post-mortem examination, the heart was found to occupy the place of the right kidney.

DEFICIENCY OF THE PERICARDIUM.

In some of the cases reported by the older anatomists as examples of congenital absence of the pericardium, it is probable that there existed pericardial adhesions, and the deficiency was only apparent, not real. But the pericardium is undoubtedly sometimes wanting. Baillie described an instance which came under his observation incidentally, the chest having been opened to explain to a class of students the normal situation of the thoracic viscera. The heart was bare, lying loose in the left cavity of the pleura. In this instance there had been no morbid symptoms referable to the heart during life. In several other examples cited by Peacock, no troubles pertaining to the heart or circulation had existed. In one instance the patient died at the age of seventy-five years with disease of the aortic valve. There are no diagnostic characters by which the existence of this abnormity can be determined during life.

MALFORMATIONS OF THE HEART.

The malformations of the heart are dependent, for the most part, on arrest of development at different periods of fœtal life. Examples of the biloculate heart, *i. e.*, the heart consisting of a single auricle and ventricle, are rare, but a considerable number of authentic cases have been reported by different observers. This abnormity is compatible with only a short duration of life, death occurring, in general, a few hours or days after birth. The pulmonic vessels in these cases are given off by the aorta, the venæ cavæ and pulmonic veins terminating in the single auricle. In this variety of malformation, the arrest of development takes place early in fœtal life. Cases in which the heart consists of only three chambers are not so rare as the preceding. They denote an arrest of development occurring at a later period of intra-uterine existence. Persons with this malformation have lived for several years, but generally death takes place within a few weeks or months. The cavities are two auricles and a single ventricle, the latter presenting sometimes a rudimentary septum. These cases differ as respects the arrangement of the primary vessels. In some cases, both the aorta and the pulmonic artery spring from the single ventricle; in others, the aorta gives origin to the pulmonary vessels, or, if the pulmonic artery exist, it is in a rudimentary form, and the blood is supplied to the lungs through the ductus arteriosus. Deficiency of the ventricular and auricular septa, either or both, is a variety of malformation vastly more common than the two varieties just noticed. When the ventricular septum is more or less deficient, the imperfection generally, but not invariably, exists at the base. The explanation of this is, the division of the cavities is here effected last during fœtal life. Hence, this form of malformation indicates an arrest of development occurring at a period still later than in the two previous forms. In the fully developed organ there exists at the upper part of the septum a triangular space in which the ventricular chambers are only separated by the endocardium and fibrous tissue on the left side, together with the lining membrane and a thin layer of muscular tissue on the right side. The average length of the sides of this triangle is about seven lines, and the base is somewhat wider. This is sometimes distinguished as the *undefended space*. It has been a question with pathologists whether, in a certain proportion of the instances of deficiency at this portion of the interventricular septum, it be not due to rupture or perforation after birth. Bouillaud contends that it is frequently attributable to disease. Peacock concurs in the opinion that it is thus attributable in some cases, but he thinks that the proportion is smaller than that claimed by the French pathologist just named. Deficiencies in both the ventricular and auricular septa are usually associated with other defects, and especially with obstruction at the pulmonic orifice. The former are probably due, in a great measure, to the latter. The effect of pulmonic obstruction on the blood-currents prevents that complete separation of the cavities (exclusive of the foramen ovale) which should take place during the latter part of fœtal life. Considerable deficiency of the partitions between the ventricles or auricles is not uniformly attended by marked symptoms referable to the heart. Persons may present few or no indications of the existence of any heart affection. If the deficiency in the septa be associated with mal-arrangement of the primary vessels, the consequences are far more serious. The pulmonic artery, as well as the aorta, may spring from the left ventricle. Virtually, in such cases, the heart consists of three cavities. The left ventricle becomes greatly enlarged, and the right ventricle proportionately atrophied. Life has continued for years under these circumstances. In other cases, the aorta, as well as the pulmonic artery, arises from the right ventricle. The pulmonic orifice in these cases is usually obstructed ; the foramen ovale remains open, and the ductus arteriosus occasionally continues pervious.

Obliteration of the pulmonic artery would at first seem to be an abnormity incompatible with life. Several cases, however, have been reported in which death did not occur for several years. In connection with this malformation, the interventricular septum is often defective. The ductus arteriosus generally remains pervious, and the pulmonary vessels are supplied through this channel. If the ventricular septum be complete, the foramen ovale continues open. The open foramen and the defect in the septum between the ventricles, instead of adding to the danger, afford relief to the overloaded right ventricle and auricle, without which life would probably not continue, except for a brief period. The right ventricle becomes greatly dilated and hypertrophied in this variety of malformation.

The presence of a supernumerary septum in the right ventricle constitutes another variety of malformation. This superfluous septum may be so far complete that the heart appears to have three ventricles. In its effects, this malformation is essentially similar to those involving obstruction or obliteration of the pulmonic orifice, and with the latter malformations it is often associated. The foramen ovale and ductus arteriosus, either or both, are generally open, and the septum between the ventricles is, in some instances, deficient. The duration of life varies according to the amount of obstruction. In the cases collected by Peacock, death occurred between the ages of nine and thirty-six years.

Certain malformations consist in the non-occurrence of those changes which should ensue after birth. The most important of these are patency of the foramen ovale and of the ductus arteriosus. These passages, peculiar and essential to the circulation in fœtal life, remain patent, one or both, after birth, in most of the instances in which the ventricular septum is more or less deficient, and, in general, their patency is associated with obstruction at or near the pulmonic orifice. When the latter condition coexists, the right ventricle generally becomes hypertrophied, but to this rule there are exceptions. If the communications between the auricles and the primary arteries be free, the right ventricle, instead of being enlarged, is sometimes found to be quite small, evidently atrophied, the blood finding a ready outlet through the fœtal passages, thus preventing accumulation within the right ventricular cavity. It is to be remarked that although in the great majority of instances of open foramen ovale, obstruction at or near the pulmonic artery is associated, the rule is not invariable. Hence, this obstruction, although a frequent, is not the sole cause of the persistence after birth of the communication between the auricles. Patency of the ductus arteriosus is also a conservative provision in cases of obliteration of, or great obstruction at the pulmonic orifice. It is equally so in certain instances in which the foramen ovale becomes closed during fœtal life. This duct remains pervious in some cases in consequence of obstruction at the aortic, and also at the mitral orifice. These coexisting malformations account for the persistence of the open duct in the great majority of cases, but the latter is sometimes observed when it is not thus to be explained, existing independently of other abnormities to which a relation of dependency can be traced.

Finally, there are certain malformations which do not interfere with the functions of the heart, but which may lay the foundations of disease in after-life. Under this head are embraced, on the one hand, deficiency, and, on the other hand, excess in the number of segments of the semilunar valves. Deficiency is of more importance than excess. In fact, it does not appear that the latter leads to any serious consequences. The former involves, in certain cases, insufficiency and regurgitation, and, probably, a disposition to take on disease greater than if the malformation did not exist. Union of the different curtains of the tricuspid valves is found not infrequently in autopsies, and in a certain proportion of these cases, may be due to disease of intra-uterine life. The proportion of cases in which the lesion dates from a period anterior to birth, is undoubtedly greater in the instances of union of the curtains of the tricuspid than of the mitral valve. It appears that the difference in tendency to valvular disease between the two sides of the heart, which is so marked after birth, is reversed during fœtal life; in other words, the tricuspid valve is as much more likely to take on disease before, as is the mitral valve after birth.

To determine in early life that malformation of some kind exists, is usually not difficult, provided the abnormity be of a nature, and sufficient in degree, to induce marked disorder. Palpitation, dyspnœa, or cyanosis, existing from birth, or developed shortly afterward, and either persisting or recurring more or less frequently, point to a congenital difficulty. To determine the particular kind of malformation, however, is a problem in diagnosis by no means always easy. With reference to the latter discrimination, it is important to bear in mind that of a given number of malformations, after the age of twelve years, in a very large proportion there exists contraction at the pulmonic orifice. Of 39 cases analyzed by Peacock, obstruction to the passage of blood into the pulmonic artery existed in 32. It is also to be kept in view that in a very large proportion of the instances in which obstruction at the pulmonic orifice exists, either there is patency of the foramen ovale, or deficiency of the auricular or ventricular septa, or both. If pulmonic obstruction be determined, the chances are that the last-mentioned malformations coexist. Guided by the law of probabilities, if a person survive several years with manifestly some cardiac malformation, we shall seldom err in presuming that there exists pulmonic obstruction. But physical signs may convert this presumption into a conclusion quite positive. A bellows-murmur referable to the pulmonic orifice points to this as the seat of an abnormal condition. We have seen that a systolic murmur may be referred to the pulmonic orifice. The maximum of the intensity of the murmur will be at the base of the heart on the left side of the sternum, or the murmur may be limited to that situation, and it is not propagated into the carotids. Attention to the pulmonic second sound of the heart may afford additional aid in the diagnosis, this sound being found to be abnormally weak or wanting. In connection with a murmur thus localized, in a large proportion of cases there will be present the physical evidence of enlargement of the right ventricle. A diastolic murmur referable to the pulmonic orifice, or a pulmonic regurgitant murmur, may be discovered, especially if the right ventricle be hypertrophied. I have met with such a murmur distinctly appreciable over the body of the heart, on the right side of the sternum, and at the xiphoid cartilage. Communication of the two ventricles through an aperture in the septum, gives rise to a systolic murmur. A murmur thus produced will not be propagated either along the course of the aorta or pulmonic artery, and will have its maximum at or near the base of the heart. By these points its source may be determined with considerable confidence, but not with positiveness, for exclusive of malformations, intraventricular murmurs are occasionally incident to disease which cannot be traced either to the arterial or auriculo-ventricular orifices by the ordinary rules of localization. The passage of blood through an open foramen ovale probably rarely, if ever, gives rise to a murmur.[1] The clinical study of cases of malformation, with respect to the physical signs, is highly interesting, and claims more attention than it has as yet received.

Of the causes of death in the various forms of malformation, the most frequent are: 1st. Cerebral disturbance resulting from the defective aeration of the blood and congestion of the brain; and, 2d. Imperfect expansion, collapse, and engorgement of the lungs. It is worthy of note that dropsical effusions, so common in lesions of the heart originating after birth, occur less frequently than would be expected from the obstruction to the circulation incident to many of the malformations. Death occurs not very infrequently from tuberculosis in the cases in which life is prolonged for several years. Of 56 cases, analyzed by Peacock, in which patients affected with different forms of malformation survived the age of eight years, in 9 tuberculosis became developed, being a ratio of 16.07 per cent. In 6 of the 9 tuberculosis cases cyanosis existed in a marked degree. This appears to militate against the incompatibility of tuberculous disease and venosity of the blood, as asserted by Rokitansky. It is, however, certain that diseases of the heart developed after birth, and phthisis are rarely associated; and the inquiry arises, whether there is a law here applicable to morbid conditions, and not to malformations. This is a question to be settled by further statistical data.

The general principles of treatment in cases of malformation, may be embraced in a very few words. They relate to measures to protect against cold, avoidance of over-exertion, and great mental excitement, together with such palliative measures as the particular circumstances in individual cases may indicate.[1]

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[1] In six cases of open foramen ovale, reported by John W. Ogle, M.D., assistant physician at St. George's Hospital, London, no murmur was discovered during life.—British Med. Jour., p. 500, 1857, from Journal de la Physiologie, &c., publié sous la direction du Docteur E. Brown-Séquard, Janvier, 1850.

CYANOSIS.

Blueness, or a purple color of the surface of the body and the mucous surfaces open to observation, occurring in connection with malformations of the heart, has been considered as constituting an affection called cyanosis,[2] morbus cæruleus, or the blue disease. For the sake of precision, these names should be restricted, as they usually are, to the peculiar coloration due to abnormal conditions which are congenital, although this effect may not be manifested for some time after birth. But an analogous, if not identical, appearance of the integument occurs, as a symptom, in cases of organic disease of the heart developed at different periods of life, and also independently of any cardiac lesions. It is well marked, for example, in the algid, or, as it is often termed, the cyanotic stage of epidemic cholera. This fact is to be borne in mind with reference to the rationale of the blueness which characterizes certain cases of cardiac malformation. The nature of the connection existing between cyanosis and malformations of the heart, has been much discussed, and is still open for discussion. To consider the subject at much length, would be inconsistent with the practical character of this work. I shall therefore present, very briefly, the views which comport best with our present knowledge.

Cyanosis was attributed by Morgagni to congestion of the venous system caused by obstruction at the origin of the pulmonic artery. John Hunter attributed it to the admixture of venous and arterial blood in consequence of an abnormal communication between the auricles or ventricles, or an abnormal arrangement of the primary vessels. The latter was the current doctrine until within the past few years the explanation of Morgagni has been revived and maintained by several distinguished pathologists—Louis and Valleix in France, Hasse and Rokitansky in Germany, Jay and Peacock in England, and Moreton Stillé of this country.[1] Many distinguished pathologists, however, still adhere to the Hunterian theory, while some adopt both explanations, referring the affection in certain cases to venous congestion solely; in other cases, to the admixture of the two kinds of blood, or to the combination of these two abnormal conditions.

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[1] For a fuller account of malformations the reader is referred to Dr. Peacock's work entitled "On Malformations, &c., of the Human Heart;" London, 1858. For an analytical review of this work by the author, see American Journal of Medical Sciences, No. for July, 1858. See, also, article "Cœur," in the Nouveau Dictionnaire, Paris, 1868.

[2] κύανος, blue, and νόσος, disease.

In the endeavor to settle upon the true explanation of cyanosis, the first and most important point of inquiry is, whether it be uniformly associated with any particular class of malformations. This point is not readily ascertained, because, in the great majority of cases, malformation does not consist of a single abnormity, but several abnormal conditions are combined. Thus, obliteration or obstruction of the pulmonic orifice generally involves an open foramen ovale or deficiency of the ventricular septum. The former induces congestion of the venous system; the latter occasions admixture of the venous and arterial blood. Analyses of large collections of cases, in fact, show that, in by far the greater number, there exist pulmonic contraction, and, at the same time, communication between either the ventricles or auricles, or both. Of 62 of the cases collated by Stillé, in which the condition of the pulmonary artery was reported, in 53 it was obstructed or impervious. In the remaining 9 cases, the author concludes that the abnormal conditions present were of a nature to give rise to congestion of the venous system. On the other hand, in 5 only out of 71 cases collected by the same author was communication between the two sides of the heart wanting. Cyanosis has been observed when the foramen ovale was not open, and there was no deficiency of the ventricular septum, nor transposition of the primary vessels; and contraction of the pulmonic orifice is not always present. Again, cases have been reported in which the two kinds of blood must have been very freely mixed, as in some instances in which there existed a single ventricle, without cyanosis; and cases of great congenital pulmonic obstruction have been observed without cyanosis.

In short, constancy of connection with any particular class of malformations has not been established. Cyanosis cannot be considered as having any fixed special anatomical character. It may be associated with numerous and different abnormal conditions.

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[1] On Cyanosis, or Morbus Cæruleus, by Moreton Stillé, M.D., American Journal of Medical Sciences, new series, vol. viii, 1844, p 25.

Continuing to regard the different forms of malformation as giving rise either to venous congestion or admixture of the two kinds of blood (although, as has been seen, both effects are usually combined), the facts adduced by Stillé, Peacock, and others, appear to show conclusively that the former effect is concerned in the production of cyanosis much oftener and to a much greater extent than the latter. The exceptions to the rule that obstruction either at the pulmonic orifice or elsewhere, inducing congestion of the veins and venous radicles, exists in cases of cyanosis, must be exceedingly infrequent, if, indeed, there be any exceptions to the rule, and the instances in which great congenital obstruction at the pulmonic orifice are not attended by cyanosis, may, perhaps, be explained, as suggested by Peacock, by supposing that the right ventricle becomes, under these circumstances, sufficiently hypertrophied to compensate for the obstruction by the increased power of its contractions. The researches of Stillé have sufficiently established the fact, already stated, that the most complete commingling of arterial and venous blood, either by direct communication between the two sides of the heart or by mal-arrangement of the vessels, is not always adequate to give rise to cyanosis; and that, as regards intensity, cyanosis bears no constant relation to the freedom of communication between the two sides of the heart or the different systems of vessels. But the establishment of these facts does not prove that the commingling of the two kinds of blood is never involved in the production of cyanosis. That in certain cases this is an important element is probable. It is evident that the coexistence of pulmonic obstruction with either an open foramen ovale or deficiency in the ventricular septum must contribute in no small measure to the admixture of the blood through these communications; and hence it is intelligible that when these malformations are combined (as they usually are), cyanosis is much more likely to be the result than when either exists independently of the other.

The general conclusions, then, most consistent with our present knowledge of the subject are that cyanosis involves, in the vast majority of cases, if not invariably, venous congestion due to contraction or obliteration of the pulmonic artery, or to some other malformation which occasions obstruction to the flow of blood from the systemic veins; that it may be produced by obstruction alone without any admixture of the arterial and the venous blood, but that the latter may contribute, more or less, to its production. The presence of venous blood in the arterial system, it is to be remarked, contributes, not alone by the purple color which it acquires from the admixture, to the cyanosis, but by increasing the venous congestion. The capillary circulation is impeded, and the flow of blood through the veins retarded in proportion to the venosity of the arterial blood.

The blueness of the skin in cyanosis is due, of course, to the blood contained in the minute or capillary vessels. Now, inasmuch as obstruction of the venous system occurs, frequently in a great degree, in cases of organic lesions of the heart arising from disease developed after birth, the question arises, why is it that cyanosis is peculiar to, or at least occurs so much oftener and to a greater extent in connection with congenital affections? It is highly probable that the answer to this inquiry is contained in a suggestion by Dr. Chevers,[1] viz., that the capillary vessels become much more largely expanded when obstruction to the circulation exists before birth, or prior to the full development of the body, the vascular system being more readily dilatable, than in the adult. Cyanotic phenomena, however, are by no means exclusively observed in connection with malformations. They may be developed at any age as a result of obstruction at the right side of the heart in conjunction with deficient aeration of the blood. They are seen in cases of pulmonary obstruction due to atelectasis, collapse of lung, capillary bronchitis, emphysema, &c. They are well marked, as already stated, in the blue stage of epidemic cholera, being dependent, in the latter affection, in a great measure, on capillary congestion proceeding from the abnormal condition of the blood itself. The appearance of the tegumentary surfaces in these various affections does not differ essentially from that in cyanosis, the main difference being that the blueness or lividity is very rarely, if ever, so extreme as in the cases in which it is dependent on congenital affections.

The discoloration in different cases of cyanosis differs greatly in degree. Between slight blueness and darkness approaching nearly to blackness, in a sufficient variety of cases, every degree of gradation will be manifested. All portions of the body are not alike affected. Certain parts, viz., the lips, around the eyes, the cheeks, the ears, the extremity of the nose, the roots of the finger nails, and the genital organs, present a change in color more marked than over the surface generally. The blueness may be limited to parts in which the skin is delicate and the capillary vessels are abundant. The degree of discoloration varies also greatly at different periods in the same case. Its intensity is increased by fits of coughing, muscular exercise, mental emotions, and any cause which excites the action of the heart. The cyanosis may exist only under these circumstances, being absent when the heart is tranquil. It is always increased by any intercurrent pulmonary or cardiac disease.

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[1] Dr. Peacock, op. cit., p. 128.

Although dependent on malformations, cyanosis is not always manifested at or immediately after birth. Of 71 cases analyzed by Stillé with respect to this point, it was congenital in 40, and occurred in the remaining 31 cases at various periods after birth. It may not occur until several years after birth. When this is the case, it is reasonable to presume that some disease of the heart or lungs has been added to the malformations, increasing the venous obstruction occasioned by the latter. It has been observed to follow a blow on the chest. The development of cyanosis after birth has been accounted for by supposing that in these cases a communication between the two sides of the heart either takes place or is enlarged at the time when the cyanosis occurs. Rupture or perforation of the foramen ovale may happen after birth, or the size of an existing aperture may be increased. The same may be said with regard to the interventricular septum at the undefended space. This explanation is based on the supposed importance of the admixture of the venous and arterial blood in the production of cyanosis. That it is applicable to certain cases is not improbable. On the other hand, cyanosis in some instances exists at birth and afterward diminishes. It may even disappear; but such cases must be extremely rare.

Although cyanosis is regarded as a distinct affection, it is sufficiently evident that it is only a symptom of certain congenital affections of the heart. It has no claim to be considered as an individual disease. It is associated with other symptoms of malformation, viz., palpitation, dyspnœa, &c. When present, habitually, in a marked degree, the patient generally is remarkably susceptible to cold, and the temperature of the body is lowered. The muscular power is deficient. The muscles do not attain to a full development. The faculties of the mind are also often imperfectly developed and feeble. Enlargement of the pulpy extremities of the fingers, with incurvation of the nails, constituting what is called "clubbed fingers," is observed in some cases. I have met with this appearance in a marked degree, in connection with organic lesions of the heart occurring after adult age, not associated with tuberculosis of the lungs.

The diagnosis rarely involves much difficulty. Discoloration of the surface, either general or partial, present habitually, or occurring whenever the action of the heart is excited; existing at, or developed shortly after birth in the great majority of instances; accompanied by palpitation, dyspnœa, tendency to syncope, &c., either constantly or in paroxysms; muscular weakness, abnormal coolness of the surface and susceptibility to cold—these are diagnostic points pertaining to the symptoms. In addition to these points, physical signs denoting malformation of the heart are generally determinable, consisting of those which denote enlargement of the organ, together with organic murmurs, the latter being often referable to the pulmonic orifice. The lividity due to certain pulmonary affections in children, is to be discriminated by the previous history, taken in connection with the presence of symptoms and signs pointing to the lungs as the seat of disease, and the absence of the symptoms and signs of malformation of the heart.

The prognosis in cases of malformation of the heart accompanied by cyanosis, is unfavorable. If the discoloration be congenital, intense, and persisting, it denotes a condition of the heart which is generally incompatible with a duration of life beyond a few weeks or months. If moderate or slight, or occurring only in paroxysms, patients sometimes live for many years, and even long life is possible. The statistics collected by Stillé with regard to the duration of the disease, show, at a glance, the diversity of cases in this respect. Of 40 cases, in all of which the cyanosis was congenital, death occurred within 23 days after birth in *seven;* between 23 days and 10 weeks, in *three;* between 10 weeks and 1 year, in *seven;* between 1 year and 10 years, in *ten;* between 10 years and 20 years, in *ten.* Of these 40 cases life was prolonged to 29 years, to 35 years, and to 57 years, respectively, in a single instance.

The treatment of cyanosis resolves itself into that of malformations of the heart. The few remarks already made comprise all that is necessary to say under this head.

CHAPTER VI.

CERTAIN AFFECTIONS INCIDENTAL TO DISEASES OF THE HEART.

COAGULATION OF FIBRIN WITHIN THE CAVITIES OF THE HEART.—Clots formed after death and at the close of life—Fibrinous coagula formed during life—Their pathological connections—Their formation in organic affections of the heart—Symptoms denoting their formation—Physical signs and diagnosis—Prognosis—Treatment.

POLYPI OF THE HEART.

ANGINA PECTORIS.—Symptoms characteristic of—Description of paroxysms—Exciting causes—Pathological character and relations—Infrequency of the affection—Influence of age and sex—Gravity and prognosis—Diagnosis—Treatment.

ENLARGEMENT OF THE THYROID BODY AND PROMINENCE OF THE EYES.—Phenomena descriptive of the prominence of the eyes—Phenomena descriptive of the enlargement of the thyroid body—Other symptomatic phenomena—Pathology—Causation—Diagnosis—Prognosis—Indications for treatment.

REDUPLICATION OF THE HEART-SOUNDS.—Different varieties of reduplication and their relative infrequency—Cases of reduplication of both sounds—Cardiac lesions found after death in cases of reduplication—Mechanism of reduplication—Bearing of the facts pertaining to reduplication on the mechanism of the normal heart-sounds—Mode of distinguishing the different varieties of reduplication—Pathological import and diagnostic significance of reduplications—Treatment.

THE caption to this chapter includes certain affections which are liable to occur in cases of organic disease of the heart, but which are not associated with any particular lesions. They occur in different forms of organic disease, and they do not, with one exception, involve, of necessity, the existence of any structural lesion. Hence, although these affections are quite dissimilar in character, they may conveniently be grouped together. First, will be considered the formation of fibrinous coagula within the cavities of the heart and the morbid growths known as polypi; second, the occurrence of pain and other symptoms in paroxysms, constituting the affection commonly known as angina pectoris; enlargement of the thyroid body and prominence of the eyeballs will be next noticed; and, finally, reduplication of the heart-sounds. These affections will be treated of only so far as they are of interest and importance in a practical point of view.

COAGULATION OF FIBRIN WITHIN THE CAVITIES OF THE HEART.

The cavities of the heart are usually found to contain, after death, coagulated blood, or clots, in more or less abundance. These are found oftener and in greater abundance in the right auricle and ventricle than in the cavities of the left side of the heart. The clots now referred to are formed post mortem. The blood remaining in the cavities coagulates after death, as it does when drawn from the vessels during life. These clots are variable as regards size, form, consistence, and color. They are sometimes uniformly dark and friable. In other instances they are more resisting, and present on the surface a layer of fibrin identical with the buffy coating of blood coagulated after venesection. The latter is sometimes tolerably firm, and, in some instances, from the imbibition of serum, it is of a soft, jelly-like consistence. A distinctive feature of the clots now referred to is, they are loose, *i. e.*, not attached to the endocardium, nor intertwined with the tendinous cords or fleshy columns. They may extend from one cavity to another through the auriculo-ventricular orifices, and into the large vessels, the arteries and veins, connected with the heart. It is not uncommon to find prolongations of considerable length contained in the large arteries, especially the pulmonary artery, consisting of fibrin, more or less solid, and colored, to a greater or less extent, by the presence of red globules. The occurrence of post-mortem clots undoubtedly depends, in a great measure, on the condition of the blood. They are formed especially after death from diseases in which the fibrinous constituent of the blood is in excess (hyperinosis); and, under these circumstances, the proportion of colorless fibrin in the clots will be increased. On the other hand, after certain fatal affections, as is well known, the blood coagulates imperfectly, and sometimes not at all, the cavities of the heart being filled with blood entirely liquid.

To the clots just described the older pathologists attached much importance. They were regarded as ante-mortem productions, and included in the class of the so-called *polypi* of the heart, being supposed to give rise to a multitude of symptoms during life, and to be frequently the cause of death. That they are formed after death is certain, but the question arises, whether they may not sometimes be formed during the last moments or hours of life, and, in fact, prove the immediate cause of the cessation of the circulation. It is difficult, and indeed impossible, to settle this question demonstratively, but the affirmative is highly probable. That coagulation of fibrin does take place before death in certain cases is not to be doubted. The clots that are indubitably of ante-mortem formation will be presently considered. The question now relates to clots, loose or unattached, and not differing from those which are due to coagulation after death. It may be readily conceived that in certain diseases of the heart, and in various affections exclusive of these, at the close of life, when the circulation becomes so enfeebled that the blood accumulates and remains nearly stagnant in the cavities, coagulation may take place, and, to quote the language of Prof. Meigs, "the last fatal blow is struck by the formation of a heart-clot of greater or less size." The author just named accounts in this way for sudden death, in some puerperal cases, during syncope induced by assuming suddenly the erect posture, when recent delivery has been accompanied or followed by a large amount of hemorrhage.[1] The explanation is, to say the least, plausible; and its extension, by the same author, to account for the arrest of the circulation in various chronic and acute diseases, is not irrational. This, however, can only be a matter for conjecture, since the clots found in the cavities of the heart do not differ from those which are formed by coagulation after death.

Masses of considerable size, consisting of coagulated fibrin, which are often found in the cavities of the heart in post-mortem examinations, furnish intrinsic evidence of having been formed during life. This evidence consists in their density, absence of red globules, intertwining with the tendinous cords and fleshy columns, adhesion to the endocardium, grooving of their surfaces by the currents of blood, and certain changes due to molecular disintegration. These characters denote that they are not produced by coagulation after death, nor as the final event in the act of dying, although they frequently prove the immediate cause of the arrest of the circulation. The date of their formation may be days and perhaps weeks anterior to death.

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[1] *Vide* paper by Prof. C. D. Meigs, in the Philadelphia Medical Examiner, March, 1849. Also treatise on Obstetrics.

These masses differ greatly in size and configuration. They may be formed in the cavities of the left, as well as in those of the right side of the heart, but more frequently in the latter. They are frequently connected with the tendinous cords and fleshy columns, with which they may be very closely and strongly intertwined. The latter fact is alone sufficient to show their ante-mortem formation. The play of the valves, when coagulation is taking place, causes the fibrin to adhere to these parts, precisely as it is collected for experimental purposes, by whipping with a bundle of small sticks blood drawn from the body. By this whipping process the red corpuscles are expelled, and the masses consist of pure fibrin. The masses are sometimes closely adherent to the endocardium, but not by means of an organized attachment. In the instances in which they seem to be grafted into the heart, the coagulated fibrin is deposited on either an exudation or a morbid growth. The opinion held by Hope, and even by some eminent pathologists of the present day, that masses of coagulated fibrin may become organized, increase by a process of growth, and undergo transformations of texture dependent on abnormal nutrition, is untenable. In the heart, as elsewhere, the fibrinous element of the blood, whenever isolated and solidified, becomes, virtually, a foreign substance incapable of organization. The fibrinous masses are sometimes found to contain collections of liquid, varying in color and consistence, presenting an appearance of unilocular or multilocular cysts. They have been said to contain pus and softened tuberculous deposit. The latter statement has not been substantiated by microscopical examinations. Either the liquid has been imbibed from without, or it is due to disintegration commencing within the solid mass; and although it may present the gross appearances of purulent or tuberculous matter, the characters of the latter as determined by the microscope are wanting.

In this account of coagulation of fibrin within the cavities of the heart, reference is had to the formation of masses of considerable size. These are to be distinguished from morbid growths and exuded lymph, the size of which may be increased by layers of fibrin, constituting the vegetations or excrescences so often found attached to the valves and orifices of the heart. The latter have been already noticed in treating of valvular lesions, and they will be again considered in connection with endocarditis. They differ from the formations now under consideration in this respect, viz., they occur almost exclusively in the cavities of the left side of the heart.

Fibrinous coagula occur in various pathological connections. They occur as a result of the accumulation and stagnation of blood in the cavities of the heart. So far, the conditions involved in their formation are mechanical. Conditions pertaining to the blood itself favor, and may be sufficient for, their formation. One of these conditions is an inordinate proportion of fibrin, either from its being positively increased, or relatively in excess owing to the other constituents of the blood being diminished. Hence, fibrinous coagula are liable to occur in certain affections characterized by hyperinosis, especially when the mechanical conditions are combined. This combination exists especially in pneumonia; and in a large proportion of the fatal cases of this disease, coagula are found which must have been formed during life.[1] It also exists in cases of death after excessive loss of fluids, as in epidemic cholera. Inflammation of the endocardium disposes to coagulation, partly from the presence of exuded lymph, and perhaps, also, from the contamination of the blood in consequence of the admixture of the inflammatory products carried into the circulation. If this latter statement be correct, it is intelligible that a similar contamination from the products of inflammation derived from other situations than the heart, may lead to the same result. The formation of fibrinous coagula is by no means limited to cases in which the heart is diseased. Some years since a heart was presented to me, in which the curtains of the tricuspid valve were literally tied firmly together by a mass of dense, colorless fibrin, portions of which were closely intertwined with the tendinous cords and papillary muscles, a prolongation of the fibrinous mass extending into the right auricle. The obstruction of the right auriculo-ventricular orifice was complete. The person had been in feeble health, but complained of no definite ailments, and was not under medical treatment. He was found dead in bed, and an examination made under the direction of the coroner disclosed no other cause of death than that stated. The heart, exclusive of the fibrinous mass mentioned, was devoid of morbid appearances; it was not enlarged, and the valves were sound.

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[1] *Vide* Traité Pratique de la Pneumonie, par Grisolle. Grisolle established by comparative observations, not only that dense fibrinous coagula, adherent or closely intertwined with the tendinous cords or fleshy columns, are often found after death in cases of pneumonia, but that they are rarely found in fatal cases of typhoid fever, peritonitis, eruptive fevers, and cerebral maladies. *Op. cit.*, 1841, p. 70 *et seq.* See, also, Richardson's prize essay On the Cause of the Coagulation of the Blood, London, 1858.

The conditions derived from organic lesions of the heart, under which fibrinous coagula are liable to occur, are mainly mechanical, consisting of the accumulation and stagnation of blood within the cavities. The latter effects, as has been seen, follow obstruction of the orifices and dilatation of the organ. In cases of mitral obstruction which has eventuated in dilatation of the right side of the heart, the time arrives, if life be sufficiently prolonged, when the right ventricle and auricle are constantly distended, the ventricular contractions being so feeble as to propel but a small quantity of blood into the engorged pulmonary vessels. Under these circumstances, the fibrin may coagulate, becoming adherent to the tendinous cords and fleshy columns, interfering with the action of the tricuspid valve, obstructing the auriculo-ventricular orifice, and prove the immediate cause of death. Or, if there exist aortic lesions involving obstruction or regurgitation, the left ventricle, after a time, reaches the limit of hypertrophic enlargement, and dilatation predominates, with consequent weakness and inability to expel but a small part of the contents of this cavity. The same result may take place here, but it occurs more infrequently than in the cavities of the right side. Fatty degeneration and extrinsic causes, which weaken the heart, together with certain states of the blood favoring coagulation, will contribute to, and may suffice for the occurrence of this accident. The mechanism of the formation of coagula, under these circumstances, is analogous to that by which they are formed in sacculated aneurisms. The formation of coagula may lead rapidly to a fatal termination. In a certain proportion of cases, sudden death is to be thus explained. This occurs when coagula become emboli and cause occlusion of either the pulmonary artery or the aorta. In other cases, life continues for some time with an aggravation of all the symptoms referable to the heart, dating from the time when the coagulation took place.

The symptoms of the formation of coagula in cases of organic disease of the heart are certainly not distinctive, but, taken in connection with all the circumstances in the case, they often point with much significance to this accident. The significance consists in a sudden and great increase of all the symptoms referable to the heart, under circumstances which render this explanation reasonable, and when the striking change in the condition of the patient is not to be otherwise accounted for. The reasoning is by exclusion rather than by positive diagnostic evidence. The formation of coagula is to be strongly suspected if a person known to have an organic affection of the heart, which has eventuated in dilatation, be abruptly seized with notable increase of dyspnœa, amounting to orthopnœa, which persists with a distressing sense of oppression at the præcordia; the heart becoming irregular and tumultuous; the pulse correspondingly disordered, extremely frequent and feeble, and the more remote symptomatic phenomena, such as lividity, dropsical effusion, coldness of the extremities, being aggravated in proportion. This suspicion is resolved into an opinion which may be entertained with great confidence, if a careful examination of the chest reveals no intercurrent pulmonary disease, nor a superadded cardiac affection adequate to account for the remarkable alteration which has ensued. An acquaintance with the symptoms and condition of the patient prior to the occurrence of the accident is important with reference to the diagnosis. If the case have not been previously under observation, the practitioner is not so well prepared to estimate properly the change as when it takes place under his own eyes. He can neither appreciate its extent nor its suddenness, and the latter is an essential point in the diagnosis. The practicability of the diagnosis presupposes that the coagula occasion obstruction to the circulation in consequence either of the space which they occupy, their situation at or near the orifices, or their interference with the proper play of the valves. A mass of fibrin, of considerable size, situated in the auricular appendix, or attached at the apex of the ventricle, may not give rise to a degree of disturbance greater than seems fairly attributable to the organic lesions, the existence of which has been ascertained. So, if the coagula are slowly formed, the symptoms are gradually developed, and the diagnosis, under these circumstances, is impracticable.

Physical signs furnish but little aid in the diagnosis. The presence of coagula may occasion an endocardial murmur, but, as a rule, it is wanting, probably in consequence of the enfeebled action of the heart. Moreover, a newly-developed murmur can hardly be discriminated from pre-existing murmurs referable to the valvular lesions which are present in the great majority of cases. Theoretically, either the tricuspid or the mitral valvular element of the first sound would be expected to be impaired or lost, according as the coagulated fibrin may impede the play of the one or the other of the auricular ventricular valves. But weakness of the heart suffices to diminish or annul the first sound. In short, the diagnostic points in cases of fibrinous coagula incidental to organic disease of the heart must, in general, be derived mainly from the symptomatic phenomena, not from physical signs.

The diagnosis of the formation of coagula in other pathological associations is free from some of the difficulties incident to their occurrence in connection with disease of the heart. For example, with the knowledge of the fact that in cases of pneumonia terminating fatally this accident is apt to occur, if, in the progress of that disease, the impulse of the heart and the pulse suddenly become extremely tumultuous, frequent, irregular, and accompanied by a degree of dyspnœa not explicable by the development of inflammation in another lobe, or any new condition referable to the lungs, the grounds for suspecting coagulation within the heart may be stronger than in some of the instances in which it occurs in cardiac affections, for the latter may have already induced great weakness and disordered action of the heart, with corresponding disturbance of respiration, &c. The change, as regards the symptoms referable to the heart, is not so striking in the latter case as in the former, and other explanations than the formation of coagula are less available. Moreover, assuming that, in the case of pneumonia, the absence of previous cardiac disease has been ascertained, if the presence of a coagulum give rise to an endocardial murmur, at the same time that the striking symptoms referable to the heart occur, this constitutes strong evidence of the nature of the accident.

The coagulation of fibrin in the cavities of the heart is a species of accident, occurring sometimes when it was not to have been looked for, and when the rationale of its occurrence is not apparent. Under these circumstances, a diagnosis may be made with considerable confidence by taking cognizance of the symptoms which the accident occasions, and by excluding other morbid conditions.

In proportion as the accumulation of the coagulated fibrin in the right auricle and ventricle interferes with the action of the tricuspid valves, and opposes an obstacle to the passage of blood through these cavities, there will be stasis in the systemic venous system, and dyspnœa. Dyspnœa is the most prominent symptom. The patient has a painful sense of the want of air, whereas it is the deficient supply of blood to be aerated in the lungs that occasions the dyspnœa. The air enters the lungs freely, and the respiratory murmur is heard on auscultation over the whole chest; but the blood, being detained in the systemic veins from the obstruction caused by the clot, is prevented from passing through the pulmonary circuit. If the obstruction be sufficient to involve impending danger, the dyspnœa is extreme. The respiration is gasping. The patient refers the seat of the oppression to the region of the heart. The stasis in the systemic veins may give rise to lividity. The surface of the body is cool or cold, and frequently covered with perspiration. The axillary temperature falls below that of health. The pulse becomes feeble, and frequently irregular, the frequency being generally, but not invariably increased. The prolonged laborious efforts of breathing and the loss of sleep induce great exhaustion, and the mode of dying is by asthenia; but toward the close of life, the patient may lapse into a semi-comatose condition.

A patient about to die from the deposit of fibrin in the right cavities of the heart is in a state not unlike that of the collapsed stage of epidemic cholera, except that dyspnœa is rarely a prominent symptom in the latter affection. The diagnosis involves, first, the absence of this, or any affection inducing the exhaustion and syncopal condition in consequence of loss of fluids, hemorrhage, or a depressing influence on the heart through sympathetic relations, or blood-poisoning. Next, diseases of the lungs are to be excluded by the absence of physical signs, together with pericarditis and structural lesions of the heart. If, in addition to these negative points, an endocardial murmur be developed which it is known did not previously exist, the diagnosis is strengthened; but the development of a murmur is not essential to the diagnosis.

Richardson states that the diagnosis may be confirmed by auscultating separately the tricuspid element of the first, and the pulmonic element of the second, sound of the heart. If the fibrin interfere materially with the play of the tricuspid valves, the valvular element of the first sound will be notably feeble or lost at the right margin of the heart; and if the clot be in the left ventricle, and impede the action of the mitral valves, the valvular element of the first sound will be weakened or lost at the apex, and heard at the right margin of the heart. The pulmonic second sound, that is, the sound heard in the second left intercostal space close to the sternum, will be weakened in proportion as the accumulation of fibrin in the right ventricle interferes with the function of the semilunar valves of the pulmonary artery. Although I have not had the opportunity of verifying these signs since reading the paper by Richardson, I can appreciate their availability. That the tricuspid and the mitral portion of the valvular element of the first sound of the heart can be interrogated separately by auscultation, is stated in another chapter of this work.[1]

These remarks on the diagnosis of fibrinous masses in the right side of the heart, occurring as an unlooked for accident, in various pathological connections, have been written directly after observing a case, the clinical facts of which apparently admitted of no other adequate explanation. It was not, however, practicable to demonstrate the correctness of the explanation by a post-mortem examination. The patient, a young married lady, arrived in this city, after a day's journey, on her way to Long Branch, and was too ill to proceed further. I saw her, in consultation with Dr. C. Y. Swan, the following day. Her health had for some time been delicate, and she had had attacks of asthma. When I first saw her, she was suffering moderately from a sense of dyspnœa, and nervous exhaustion, attributable to fatigue and loss of sleep. There was notable hyperæsthesia of the surface of the body. On an examination of the chest, the respiratory murmur was found to be everywhere normal, and there were no cardiac signs of disease, except a reduplication of the second sound of the heart. The symptoms in the case were considered as denoting only disorder of the nervous system. Four days afterward, I saw the patient again. In the meantime, the dyspnœa had increased, and she had obtained very little sleep. The suffering now from a sense of the want of breath was very distressing. The pulse was extremely small and feeble, numbering, however, only 80 per minute. The surface of the body was cold, and covered with perspiration. The prolabia were livid. The mental faculties were intact. She had no vomiting, nor diarrhœa, nor aught else to account for the collapsed condition. A careful examination of the chest showed everywhere resonance on percussion, and a normal respiratory murmur. There was no cardiac murmur, and the reduplication of the second sound had ceased. Eight hours afterwards, I found the patient moribund, the pulse being extinct at the wrist. At this visit, for the first time, I discovered a systolic murmur over the heart. The murmur was limited to the præcordia, being most marked between the base and apex. This was an hour only before death.

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[1] *Vide* page 68. For paper on the Diagnosis of Polypi of the Heart, by Benjamin W. Richardson, M.D , see London Med. Times and Gazette, November 21, 1868, and the Half-Yearly Abstract of the Medical Sciences, reprinted by Henry C. Lea, No. for July, 1869.

In the foregoing remarks on the diagnosis, I have had reference chiefly to the formation of fibrinous clots in the right cavities of the heart. The accumulation of fibrin in the left cavities gives rise to symptoms which are less distinctive. Pulmonary congestion occurs in proportion as the accumulation in these cavities opposes an obstacle to the return of blood from the lungs. The dyspnœa which exists is mainly attributable to this congestion. The action of the heart is tumultuous and irregular. The pulse is small in proportion as the quantity of blood in the left cavities is deficient. The occurrence of fibrinous clots in these cavities is extremely rare, exclusive of the cases in which it is incidental to structural affections of the heart involving weakness of the left ventricle from either dilatation or fatty degeneration.

The prognosis in cases in which the symptoms denote the formation of fibrinous coagula is in the highest degree unfavorable. Bouillaud entertains the belief that the coagula are sometimes dissolved and disappear. The ground for such a belief is so small as to render it excusable to conclude that, in the instances in which this favorable termination has been supposed to take place, an error of diagnosis may have been committed. As incidental to organic affections of the heart, however, it is to be borne in mind that in most instances the condition of the patient prior to this accident was hopeless. The effect of the latter, in general, is only to hasten the period of relief from the sufferings incident to incurable disease.

As regards treatment, it follows from the remarks just made that, after coagula have formed, palliative measures are alone indicated. These consist of remedies to relieve dyspnœa and præcordial distress, stimulants to maintain the action of the heart, and revulsive applications such as fomentations, sinapisms, and stimulating pediluvia. The idea of giving remedies with a view to dissolve the solidified fibrin is absurd. To prevent the coagulation of fibrin, when circumstances are present under which it may be expected to occur, is a legitimate, and may be an important object of treatment. This object involves, in the first place, obviating as far as possible the accumulation of blood in the cavities of the heart by measures which have been already considered in connection with the treatment of valvular lesions. Sedative remedies, pushed to the extent of retarding and weakening unduly the muscular contractions of the heart, are objectionable, among other reasons, on the ground that they may favor coagulation. Digitalis is by some writers regarded as a dangerous remedy on this ground in cases of advanced organic or other disease in which the organ is already enfeebled. This remedy, however, is unattended by danger, if it be true that, while it retards the movements of the heart, it does not diminish the muscular power of the organ. In the second place, it is not improbable that certain remedies may favor the liquidity of fibrin in the blood, in this way preventing coagulation. Alkaline remedies have been supposed to have this effect. If it be true that the fibrin is in a liquid form in the blood from the presence of ammonia, it is a rational inference that ammoniacal remedies must be the most efficient in fulfilling this second object in the prophylactic treatment.

POLYPI OF THE HEART.

The clots and fibrinous coagula which have been considered, were regarded, as already stated, by the older pathologists as morbid growths resembling the polypi met with in the uterus, nasal passages, and other situations. They were called *polypi* of the heart, and the term *polypoid* formations is still very generally applied to them. It is needless to say that, pathologically, they bear no resemblance to polypi, since they are not morbid growths, and never become organized. They cannot, therefore, with propriety be said to be *polypoid*, and the use of this term has been designedly avoided in the foregoing remarks. Abnormal productions, however, may occur within the cavities of the heart, which are analogous to polypi or polypoid growths. Grisolle[1] analyzed seven cases which he stated to be all the authentic cases then on record.[1] In nearly all these cases there existed a pedunculated tumor varying in size from a pigeon's egg to a hen's egg; in six of the cases it was contained in the right, and in one case in the left auricle. In all the cases in which the point of attachment was indicated, it was at or near the foramen ovale. In four cases the tumor extended through the auriculo-ventricular orifice into the ventricle. The peduncle was formed apparently by the endocardial membrane which generally extended over the tumor. The form of the polypi was pyramidal, and they presented in some cases a smooth, and in other cases a lobulated surface. The substance of the tumors varied in appearance. In one case it had a fleshy aspect, in one case it resembled a fungous growth, and in two cases the texture was fibrous. In every case there was hypertrophy of the auricle and the corresponding ventricle.

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[1] Traité de Pathologie Interne, 1852, t. ii, p. 389.

A single example only of polypoid tumor within the heart has fallen under my observation. The following is an account of the case, from the records of autopsies at Bellevue Hospital:

An unknown man, aged about 35, was found in the street, December 29th, 1866, as supposed, in a state of intoxication. He was locked in a cell of a police station-house, and was afterward found dead. The post-mortem examination was made by Dr. Wooster Beach, Jr. The brain appeared to be healthy; the pericardium was distended with liquid, so that it occupied a large portion of the thorax; the lungs were adherent to the pericardium and the costal pleuræ, otherwise they were normal; the kidneys appeared to be fatty; the liver was in an advanced stage of cirrhosis.

The surface of the heart was covered with recent inflammatory exudation. The left auricle and the mitral valves were normal. The left ventricle was of normal size. The aortic valves were slightly atheromatous. A moderate quantity of dark coagulated blood was contained in the left auricle and the left ventricle. The right ventricle was much dilated. The pulmonic valves were normal. The tricuspid orifice was very large, and the valves were folded back and adherent to the ventricular wall. The valves were thickened and roughened. The right auricle was much dilated. Attached to the inner surface of this auricle, about half an inch from the auriculo-ventricular opening, were two polypi. One, nearly spheroidal in form, two and a half inches in its greatest diameter, having a very small pedicle, was formed of a yellowish, translucent, elastic tissue, with one or two spots of calcareous degeneration. This polypus was contained entirely within the auricle. The other, situated close to the first, was three and a half inches in length, ovoid in shape, and was mostly calcareous. The lower end presented an appearance as if a portion had been broken off. It protruded through the auriculo-ventricular opening into the ventricle.

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[1] The museum of the Boston Society for Medical Improvement contains a specimen presenting a tumor hanging loose in the cavity of the left auricle, supposed to be malignant from the coexistence of malignant disease in the lung and about the elbow. *Vide* printed catalogue, 1847, p. 88.

On the surface of both, vessels filled with blood were seen with the naked eye by several observers. A thin membrane was dissected up from the surface, which consisted of a delicate lacework of fibres, with a few spindle cells. The mass of the tumors consisted of, 1st, bloodvessels, some of which contained blood; 2d, cells, spindle-shaped, star-shaped, round, and oval, .018 m.m. in diameter, most of them being somewhat fatty; 3d, fibres in broad bands, and 4th, basement membrane, transparent, homogeneous, and finely granular.

The left primary branch of the pulmonary artery was plugged with a large calcareous mass. The arterial coats were thinned as if the mass had been in this situation for some time.

The formation of true polypi in the heart differs from that of clots and fibrinous coagula in this, viz., it goes on slowly, and a considerable period must be required for the growth of a tumor of sufficient size to occasion serious inconvenience. Hence, the symptoms are developed gradually and imperceptibly, not abruptly as in cases of coagula. When developed, the symptoms denote an organic affection of the heart, without pointing to the existence of a tumor. The physical signs, as well as symptoms, are not distinctive of the nature of the affection. They may indicate obstruction, or regurgitation, or both, associated with more or less cardiac enlargement. Taking into view the excessive infrequency of these growths, their existence can hardly be suspected from the phenomena during life.

It is needless to consider the treatment. The fact of some obscure cardiac affection being determined, the indications will be derived from the condition of the heart as respects enlargement, and the symptoms.

ANGINA PECTORIS.

An extremely distressing and grave affection, occurring, happily, in a very small proportion of cases of organic disease of heart, is commonly known by the name angina pectoris.[1] This name was applied to the affection by Heberden, who was the first to give a full and clear description of it in 1768. The distinctive features of the affection were portrayed in connection with the report of a case, in the same year, by a French writer, Rougnon. The affection is characterized by paroxysms of intense pain emanating from the neighborhood of the præcordia, extending thence in various directions, often into the left shoulder and down the arm, accompanied by indescribable anguish, a sense of suffocation, and a feeling of impending death. These are symptoms characteristic of the affection in a severe form. The præcordial pain is variously described by patients, as lancinating, contusive, lacerating, burning, or constrictive. Its centre or focus is generally over the heart, to the left of the sternum. It is sometimes most intense beneath the sternum; and Valleix cites two instances in which the greatest intensity was referred to the right of the sternum.[2] The pain radiates into both sides of the chest, into the back, extending, as already stated, often into the left upper extremity, sometimes into both upper extremities, occasionally into one or both of the lower extremities, and in rare cases it pervades all the extremities. Not uncommonly it ascends to the neck in front or behind, and it may extend to the jaws and temples. The pain in the upper extremity sometimes appears to end abruptly at the shoulder, and in other cases at the elbow. I have known it to be felt acutely in the forearm, and not in the arm or shoulder. Not infrequently it seems to follow the course of the nerves, and is felt over the whole affected extremity, even to the fingers. As a very rare exception to the rule, the pain sometimes extends exclusively to the right upper extremity. The pain is attended by a feeling of numbness, or as if the limb were paralyzed. Numbness with pain, referred to the testes is a rare concomitant. Tenderness to the touch, or hyperæsthesia of the integument over the situations in which the pain is felt, has been observed in some cases, especially in females.

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[1] ἄγχω, to strangle.

[2] Guide de Médecine Pratique.

The affection is essentially paroxysmal. The patient is seized suddenly, often when in motion; and the paroxysm has been repeatedly noticed to occur in walking up an acclivity, after a meal, and especially against a strong current of air. In severe cases instant immobility is imperative. He seizes hold of some firm support, if any be at hand, or he finds it necessary to take a sitting or recumbent posture, which he does with great caution, and remains in a fixed position until the paroxysm passes off. The pain in certain cases is by no means the sole element of the distress. A sense of suffocation and of impending dissolution occasions hardly less suffering. There is, in addition, a feeling of anguish which patients find it impossible to describe. Dyspnœa is not an essential element of the paroxysms. The respiratory movements are often suspended for an instant, or restrained by an act of the will, from fear of increasing the pain and distress, but the ability to expand the chest and breathe regularly is not impaired. Relief is sometimes obtained by holding the breath. For fear of increasing the distress, the patient avoids speaking as much as possible. Not infrequently the respirations continue unaffected during the paroxysms, and they are rarely more than moderately accelerated. Palpitation is often present. The action of the heart is frequently irregular and intermitting, the pulse indicating sometimes vigor and sometimes feebleness of the ventricular contractions. The pulse is sometimes unnaturally slow during the paroxysms. The countenance is pallid, and expresses anxiety, terror, and distress. The change in this respect may be very striking, a deathlike complexion, with great haggardness of the features, suddenly taking the place of an appearance of health. Lividity is occasionally observed. The surface is cold, and frequently bathed in perspiration. The faculties of the mind remain unaffected. A free secretion of limpid urine takes place in some cases. The reader who has not witnessed a severe paroxysm, may form from the foregoing account some idea of its distressing nature. There are few, if any, diseases which give rise to greater suffering. A patient who experienced the excruciating torture of daily attacks for several months before he found relief in death, made a dying request that I should examine his body post mortem, in the hope that something might be thereby ascertained which would lead to the means of relieving others in like manner afflicted; a request with which I did not fail to comply.

The paroxysms of angina, in different cases, differ much in severity, as well as in frequency of recurrence, duration, &c. They by no means always have the severe character which has been portrayed in the foregoing sketch. They are sometimes comparatively mild. The affection in some cases commences with mild paroxysms which may progressively become more severe; but in other cases the first attack is intensely distressing. Their duration is extremely variable. Often they last only for a few moments or even seconds, but in some of the cases which have fallen under my observation, the suffering has continued for several hours. The cessation of the paroxysm is generally as abrupt as the commencement. I have known a laborer to be attacked repeatedly while at work, and, resting for a few moments till the paroxysm ceased, at once to resume his labor. In some cases, however, relief is gradual. There exists great diversity in different cases, as respects recurrence of the paroxysms. They may recur after intervals of a few hours, or even moments; or days, weeks, months, and years may elapse between successive attacks. The paroxysms are apt progressively to become more and more frequent, as well as more and more severe. Patients sometimes have repetitions, more or less frequent, during several days or weeks, and a respite for several months follows. As will be seen presently, the attacks, when not associated with disease of the heart or aorta, may cease to recur, the immunity from the affection being permanent; but when associated with disease of the heart or aorta, the attacks, in some cases, having been more or less frequent for a certain period, do not afterward recur. In a case under my observation, the patient had repeated attacks during a period of two months, when they ceased and did not return, death taking place several months afterward. In another case, two extremely severe paroxysms occurred, the interval being about forty-eight hours, each paroxysm lasting from three to four hours, and the patient, aged sixty-seven, never had a recurrence, dying, after ten or twelve years, with cardiac disease.

The paroxysms of angina are frequently induced by some obvious exciting cause, such as ascending an elevation, muscular exertion of any kind, mental excitement, &c. But in many cases they occur without any such cause, taking place in the night, or when the person affected is perfectly at rest in body and mind. When they recur with great frequency, slight causes seem adequate to determine an attack. I have known the act of mastication sufficient, so that the patient resisted as long as possible the desire to take solid food. In the same case the paroxysms often occurred during sleep, appearing to the patient to be produced in consequence of dreams. On account of this liability, he hardly dared to sleep. The suffering in this case was beyond description. In some of the cases which I have observed, the paroxysms occurred only when it seemed fair to refer them to some obvious exciting cause. In one of my cases the paroxysms always occurred, as it were, spontaneously, and causes which provoked severe attacks of dyspnœa, or cardiac asthma, never occasioned angina.

We come now to inquire what is the pathological character of this affection, and what are its pathological relations. It is sufficiently clear that the affection is a form of neuralgia. The various kinds of pain are those which belong to neuralgic affections. The radiation of the pain in different directions is characteristic of the latter. Moreover, in many cases, the pain distinctly follows the course of the nerves of the extremities. The identity is further shown by the abruptness of the attack, the suddenness with which the paroxysm frequently ends, and the completeness with which it disappears, leaving no trace of the affection except a certain amount of soreness and prostration. Heberden and others have attributed the pain to spasm; but a spasmodic contraction of the heart sufficient to occasion such prolonged as well as intense suffering would be incompatible with life. Disturbance of the action of the heart is by no means, as a rule, proportionate to the amount of pain, and it is not a constant element of the paroxysms; the pain in some cases is extreme, while the movements of the heart are but little or not at all disturbed. Disordered action often constitutes an element of the paroxysms of angina, but that this element is distinct from the neuralgia, is shown by the fact that functional disturbance of the heart, be it never so great, is rarely accompanied by pain. Neuralgic pain, and disturbed muscular action of the heart, are thus two elements of angina, which although associated, are pathologically distinct, and the latter may be entirely wanting. The term *cardialgia*, had it not been appropriated to denote pain supposed to be referable to the cardiac extremity of the stomach, would be a more appropriate name than angina, for the affection under consideration. This term was applied by Forget to pain seated in the heart.[1]

As regards the pathological relations of angina, it involves, in a large proportion of cases, the existence of some organic affection of the heart or aorta. Does it involve the existence of any particular kind of lesion? In answer to this question, dissections show that the lesions found in different cases do not agree, invariably, in any appreciable morbid alterations. The heart may or may not be enlarged. Valvular lesions are either present or wanting. Calcification of the coronary arteries is the only appreciable lesion in some cases, but in other cases these arteries are found to be entirely healthy and free from obstruction. Fatty degeneration is sometimes observed, but by no means as a rule. In the two most severe cases that have fallen under my observation, the substance of the heart presented no appearance of fatty change. The affection undoubtedly occurs oftener in connection with lesions seated at the aortic orifice or in the aorta than elsewhere. But that lesions here are not essential is sufficiently established. Doubtless some particular abnormal condition is common to all the cases of organic disease of the heart in which angina occurs, but its nature and seat are yet to be determined. With our present knowledge we have nothing but conjectures on this point. There will be no advantage, in a practical point of view, in considering these. I will simply remark that the hypothesis which attributes the occurrence of angina to simple weakness of the heart, has no better foundation than that which attributes it to spasm. Not only is the heart weakened in a host of cases of dilatation and fatty degeneration, without the occurrence of angina, but weakness by no means exists uniformly in the cases in which angina occurs. In several of the instances which have come under my observation, a paroxysm of angina was the first event to denote the existence of cardiac disease, and patients continued for a long time to take active exercise, performing, for example, severe manual labor, without inconvenience save from the recurrence of angina. In two of the severest cases which I have observed, the patients, even a few weeks before death, were confident of their ability still to work, if the frequent attacks could be prevented. These facts are inconsistent with much weakness of the heart. Moreover, the physical signs in certain cases, for a longer or shorter period, show that the heart acts with vigor, and, in some cases, with the abnormal power incident to hypertrophy.

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[1] Op. cit.

Angina pectoris is not always accompanied by the evidence of lesions of the heart or aorta. There is reason to believe that it may be a purely functional affection. This statement is not in accordance with views held by some writers, and it is opposed to those which were adopted in the first edition of this work. Heretofore I have considered true angina as invariably incidental to cardiac or aortic disease, considering all cases in which more or less of the symptomatic phenomena are manifested without the signs of disease of the heart or aorta, as cases of simulated or pseudo angina. I am satisfied that this doctrine is incorrect, inasmuch as the distinctive features of the affection may be as well marked without, as with, these signs. In its practical bearings the question whether well-marked angina may exist without being connected with cardiac or aortic lesions is of much importance. Assuming this connection to be invariable, the angina, of course, is to be taken as sufficient evidence in itself of disease of the heart or aorta by those who do not rely upon physical signs. The question is of great importance with regard to prognosis. There is abundant room for patients to hope for permanent immunity from the affection if it be not connected with the disease of the heart; whereas, the ground for such a hope is much less if the affection be incidental to lesions. Moreover, the danger of sudden death, which is considerable if the affection be associated with disease of the heart, is much less if the heart be free from disease. In view of the importance of the question in these aspects, I shall subjoin a brief account of several cases which I have recorded.

Case 1. Prof. R., aged 40, an eminent member of the medical profession, consulted me in September, 1868. Nine years previously he had repeated attacks of hæmoptysis, together with cough and expectoration, diarrhœa, and emaciation. He recovered after a year. He was afterward subject to asthma, but of late years he has had attacks of this disease only when he inhales the powder of ipecacuanha. The smallest quantity of this drug, will excite an attack. At intervals of about four months during the four years preceding the date of his consulting me, he had had paroxysms of angina pectoris. The pain was excruciating, shooting into the left arm, and accompanied by a vivid sense of impending death. The paroxysms lasted about a minute. During their continuance he was compelled to remain immovable. His weight was 184 pounds. He presented an aspect of health, and was actively engaged in medical practice; he suffered, however, from dyspeptic ailments. On examination of the chest I failed to find any signs of valvular lesions, or of enlargement of the heart.

Prof. R. consulted me again in July, 1869. After the date of my former examination he had no return of the angina for about seven months. He then had two severe paroxysms following each other after an interval of a few minutes. Afterward for several weeks he had slight paroxysms with intervals of three or four days. A second physical examination was negative as regards signs of disease of the heart or aorta. His general health was excellent. He had been very actively at work in practice, and was on his way to Europe for medical observation and recreation. In this case, during the severe paroxysms just referred to, the pulse fell to 40 per minute, and became very feeble.

CASE 2. Mr. H., aged 56, farmer, consulted me July 27, 1865. He had been subject for several years to dyspeptic ailments. During several weeks prior to the date of the consultation he had had repeated attacks of pain in the præcordia extending into the left upper extremities. The attacks were brought on by either muscular exercise or mental excitement. They were not accompanied by palpitation. On examination of the chest, no signs of disease of the heart were discoverable. His aspect was healthy. He had taken a variety of remedies, and for some time had been on a reduced diet. He had been a very active hard-working man. Recreation, the long-continued use of tonic remedies, varied from time to time, and a good diet were advised. Nearly three years afterward I learned that this patient was in excellent health.

CASE 3. Dr. H., aged 35, consulted me March 8, 1865. For the preceding five years he had been subject to paroxysms of pain in the præcordia, shooting in different directions, sometimes into the right, but never into the left upper extremity, accompanied by a sensation as if the heart were compressed. He described the suffering as a "heart-pang." The paroxysms occurred suddenly, lasting from half a minute to an hour, and relief was obtained by making strong pressure on the præcordia. During the period stated, namely five years, he had sometimes been exempt feom the paroxysms for several successive months, and at times they occurred repeatedly during the day for successive days. For several months, under a conviction that these paroxysms denoted disease of the heart and a liability to sudden death, he had relinquished his practice and kept the house most of the time. He had no palpitation either with the paroxysms or at any other time. An examination of the chest was negative as regards any signs of disease. Mental recreation was advised, with assurances of the non-existence of disease of the heart. In a letter received about two years afterward, Dr. H. stated that he was in good health and actively engaged in the practice of medicine.

Case 4. January 29, 1869, I visited, in consultation with Dr. Robert F. Weir, Mrs. B., aged about 60. For several months she had been subject to attacks having the characters of angina pectoris. On examination of the chest, no cardiac murmur was found and no signs of enlargement or of fatty degeneration. On the absence of physical evidence of cardiac disease, a favorable prognosis was given. The patient, however, in the following April died suddenly in a paroxysm. On examination after death the heart was found to be not enlarged, and its structure was healthy. The aorta was somewhat dilated, and there were atheromatous and calcareous patches above the valves. The valves, however, were sound. A calcareous mass existed at the base of one of the mitral curtains, but the latter were sound. The condition of the coronary arteries is not noted, but I cannot doubt that they were examined and nothing abnormal found.

In this case the heart was not entirely devoid of lesions, but they did not give rise to physical signs, and, as regards immediate morbid effects, they were innocuous. The sudden death was certainly not attributable to the lesions. This case, it must be confessed, gives support to the surmise that, in other cases in which no signs of disease of the heart or aorta are discoverable, lesions nevertheless may exist. Assuming the surmise to be correct, the question, as in this case, is an open one whether the lesions have aught to do with the development of the angina, or whether the association is one merely of coincidence.

Reverting now to the question, what is the pathological character of this affection, and what are its pathological relations, it is a neuralgic affection very often, but not invariably, incidental to lesions of the heart or aorta. The frequency of its association with these lesions shows some pathological connection, and the connection involves some unknown element which is common to different lesions. It may be associated with lesions which appear to be trivial, as well as with those which are grave. Its occurrence in a certain proportion of cases, without being associated with lesions, of course, disposes of the question whether it is to be regarded merely as a symptom and not as a distinct affection. Moreover, the striking and distinctive phenomena which it embraces are sufficient grounds for regarding it as an individual disease.

Assuming angina pectoris to be a neuralgic affection, in what nerves is it seated?

That the paroxysms involve, as a point of departure, neuralgia of the cardiac nerves is a fair inference from the constancy of the præcordial pain. That the disturbed action of the heart in certain cases is due to an affection of the pneumogastrics, is probable in view of the knowledge of the function of these nerves, as regards the heart, obtained, within late years, by experimental researches. Galvanizing or irritating the divided extremities of these nerves in the neck retards, renders unequal and irregular, and finally arrests, by paralysis, the action of the heart. It may be reasonably supposed that, in certain of the paroxysms of angina, the same effects are produced by means of a morbid condition of the pneumogastric nerves. The neuralgic affection, however, extends to other than the cardiac nerves in the great majority of cases. Generally it extends to the brachial plexus and the nervous trunks of the left upper extremity. It may extend to the same nerves of the right upper extremity, to the nerves of the lower extremities, to the intercostal nerves, &c. These extensions of the neuralgia to other nerves are due either to a successive invasion of different nervous trunks, or to a reflex effect of excitation transmitted by the pneumogastrics to the nervous centre.

Although the affection is essentially neuralgic, more or less disturbance of the heart's action is generally associated with the neuralgia. The disturbance of the heart's action explains certain symptoms which frequently enter into severe paroxysms, namely, the sense of impending dissolution and the necessity of maintaining immobility of position. The danger which will presently be considered depends on the disturbance of the heart's action. This disturbance is represented by the pulse, as well as by the signs obtained by palpation and auscultation. Of course, the disturbance of the heart's action is greater, more distressing, and more serious, other things being equal, in the cases in which the affection is incidental to cardiac lesions, and in proportion to the gravity of the latter. But disturbance of the heart's action, although a frequent, is not a constant element of the paroxysms of angina. It is a superadded element which in a certain proportion of cases is wanting.

Angina pectoris is a rare affection. Of over one hundred and fifty cases of organic disease of the heart, as evidenced either by the results of examination after death or well-marked physical signs, the histories of which were before me when this work was written, in 1859, this intercurrent affection existed in seven only. It existed in only 8 of 188 cases entering into the collection analyzed in preparing a second edition (1869). It is worthy of note that the ratio of its occurrence is so nearly the same in the two collections of cases. Adding together the two collections the proportion is 15 in 338 cases. This infrequency shows that it depends on conditions which, so far from being associated with structural lesions as a rule, are quite exceptional. Its connection with organic disease of the heart might be considered as merely a coincidence, were it not that it occurs so much oftener with, than without, this connection. During the past ten years (1869) I have noted only four cases of angina not connected with cardiac or aortic lesions, their exclusion, in all the cases, being based on the absence of physical signs. An account of these cases has been introduced.[1] In one of the cases in which the heart was not found on examination after death to be entirely free from disease (case of Mrs. B., introduced with the three cases just referred to), there were no signs of cardiac lesion. In another case, which will be presently introduced, the only physical sign was an aortic direct murmur, the aortic valves being sound, and the heart not enlarged. In the remaining six cases, in which cardiac disease existed, in five there were present signs denoting aortic insufficiency. In one case there was a murmur at the apex, none existing at the base, and there was physical evidence of fatty degeneration of the heart. In one case, in connection and aside from certain exciting causes, such as excessive mental emotions, which are involved in only a certain proportion of cases, our knowledge embraces but little more than the influence of sex and age. The affection occurs much oftener in males than in females. Of 88 cases analyzed with reference to this point by Forbes, 80 were males, and 8 females. This immense disproportion shows that a very decided causative influence pertains to sex. A similar influence belongs to age. Of these 88 cases in 72, the age exceeded fifty years. I have met with the affection well marked in a female of twenty years of age, who subsequently died suddenly during an attack. Cases in which it has occurred in childhood, and even in infancy, have been observed.

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[1] *Vide* page 293.

With reference to the prognosis in cases of angina pectoris, it is important to endeavor to determine, in individual cases, whether the affection be purely functional, or whether it be associated with aortic or cardiac lesions. The exclusion of lesions is to be based on the absence of their physical signs. If no evidence of aneurism, atheroma, or calcification of the aorta be discoverable; if the heart be not enlarged; if fatty degeneration be not evident, and if organic cardiac murmur be wanting, it may be presumed that the affection is functional. The absence of the physical signs of aortic and cardiac lesions, however, does not suffice for their positive exclusion. Aneurism, atheroma, and calcification of the aorta are not always manifested by signs; a certain amount of fatty degeneration may exist without being discoverable; obesity of the heart has no physical manifestations; calcareous deposits within the cavities do not invariably give rise to murmur, and obstruction of the coronary arteries is beyond the reach of physical diagnosis. Although, therefore, the result of an examination of the chest be negative, there is room for the supposition that lesions may exist, and that the angina is dependent upon them. The prognosis, with reference to the persistence of the affection, is favorable in proportion as there is reason to believe that the affection is functional. After the occurrence of veritable paroxysms of angina during a greater or less period, permanent exemption from their recurrence may be expected if the affection be not connected with lesions.

The paroxysms of angina involve a liability to sudden death, the most rational explanation being, that the action of the heart with an aortic direct and an aortic regurgitant murmur, there was a mitral direct murmur; and in this case the existence of mitral contraction was verified by a post-mortem examination. In another case, a mitral direct murmur existed with the same aortic murmurs as in the foregoing case, the patient not dying while under my observation. Of all the cases which I have recorded during the last ten years, 12 in number, a fatal termination is noted in only three. Exclusive, however, of the three fatal cases and of three cases among those introduced, the patients were lost sight of either after a single examination or after a brief period of observation.

Regarding angina pectoris as an affection dependent generally, but not invariably, on some causative agency common to different lesions seated in the heart or aorta, the question arises, what are its causes when it occurs independently of these lesions, that is, when, with our present knowledge, it is to be considered as a purely functional or an idiopathic affection? This question cannot be answered satisfactorily. The affection is supposed to be one of the manifestations of rheumatism and gout. But there seems to be not much ground for this supposition. Of the cases of rheumatism and gout in which the heart and aorta are free from lesions, angina occurs in an exceedingly small proportion; and, on the other hand, functional angina in the majority of cases is not associated with the unequivocal rheumatic or gouty phenomena. In none of the cases of angina, without the signs of cardiac or aortic lesions, which have come under my observation, had the patients been affected with either rheumatism or gout. The most that can be said is, that rheumatism and gout may act as auxiliary causes. The same statement is applicable to another causative agency, namely, dyspepsia. Dyspeptic ailments exist in a certain proportion of the cases of functional angina; but the association is not invariable, and how common are these ailments without angina! This mode of reasoning renders untenable the opinion of Trousseau, who considered angina as a form of epilepsy. The concurrence of these two affections is not frequent enough to prove anything beyond coincidence; and the points of resemblance, namely, the sudden access, together with the equally sudden termination of the paroxysms, and generally, their brief duration, are certainly quite
establish the identity of the affections. In short
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and aside from certain exciting causes, such as excessive mental emotions, which are involved in only a certain proportion of cases, our knowledge embraces but little more than the influence of sex and age. The affection occurs much oftener in males than in females. Of 88 cases analyzed with reference to this point by Forbes, 80 were males, and 8 females. This immense disproportion shows that a very decided causative influence pertains to sex. A similar influence belongs to age. Of these 88 cases in 72, the age exceeded fifty years. I have met with the affection well marked in a female of twenty years of age, who subsequently died suddenly during an attack. Cases in which it has occurred in childhood, and even in infancy, have been observed.

With reference to the prognosis in cases of angina pectoris, it is important to endeavor to determine, in individual cases, whether the affection be purely functional, or whether it be associated with aortic or cardiac lesions. The exclusion of lesions is to be based on the absence of their physical signs. If no evidence of aneurism, atheroma, or calcification of the aorta be discoverable; if the heart be not enlarged; if fatty degeneration be not evident, and if organic cardiac murmur be wanting, it may be presumed that the affection is functional. The absence of the physical signs of aortic and cardiac lesions, however, does not suffice for their positive exclusion. Aneurism, atheroma, and calcification of the aorta are not always manifested by signs; a certain amount of fatty degeneration may exist without being discoverable; obesity of the heart has no physical manifestations; calcareous deposits within the cavities do not invariably give rise to murmur, and obstruction of the coronary arteries is beyond the reach of physical diagnosis. Although, therefore, the result of an examination of the chest be negative, there is room for the supposition that lesions may exist, and that the angina is dependent upon them. The prognosis, with reference to the persistence of the affection, is favorable in proportion as there is reason to believe that the affection is functional. After the occurrence of veritable paroxysms of angina during a greater or less period, permanent exemption from their recurrence may be expected if the affection be not connected with lesions.

The paroxysms of angina involve a liability to sudden death, the most rational explanation being, that the action of the heart is arrested by a morbid agency affecting it through the pneumogastric nerves in the manner in which irritation of these nerves, or the electrical current produces this effect in experimental observations. The danger is great in proportion as the action of the heart is irregular, feeble, or retarded during a paroxysm. Per contra, if during a paroxysm the action of the heart be but little or not at all disturbed, the patient may be considered as free from the liability to sudden death. The prognosis, as regards sudden death, is favorable if the affection be functional, or if existing lesions do not, in themselves, involve danger. The physician, however, is not warranted in saying that there is no liability to sudden death under these circumstances. I have cited a case from among those which have come under my observation, in which sudden death occurred during a paroxysm, the lesions found after death evidently having nothing to do with the fatal termination, except, perhaps, as entering into the etiology of the angina. On the other hand, the liability to sudden death is much increased by the existence of serious lesions, especially those occasioning free aortic regurgitation, and also notable dilatation and fatty degeneration of the heart. It is easy to understand the additional danger incident to these lesions in a paroxysm of angina in which the action of the heart is much affected. The danger, under these circumstances, will be proportionate to the extent to which the heart is weakened by lesions, together with the amount of disturbance of the heart's action incident to the angina.

Aside from the liability to sudden death, the importance of angina, as regards prognosis, is in proportion to the severity of the paroxysms, and the frequency of their recurrence. Severe and frequently recurring paroxysms of angina interfere with the tolerance of aortic or cardiac lesions when these are associated, and thus shorten the duration of life; in other words, lesions in themselves more or less serious are rendered more so by the association with angina, in proportion as the latter is severe. But it is to be borne in mind that the paroxysms of angina sometimes cease to recur in cases in which it is incidental to serious lesions. The prospect of immunity from the paroxysms of angina is greater when the lesions are innocuous than when they are serious. The following case illustrates notable improvement, the only evidence of lesions being an aortic direct murmur:

Rev. Mr. D. consulted me in March, 1868. He was sixty years of age, healthy in appearance, and up to his present illness had always been well. In the preceding November he began to have attacks of præcordial pain excited by walking in the open air. They had recurred at short intervals, and, of late, had been almost invariably excited by any unusual physical exertion. They had also sometimes occurred when he was entirely at rest; the pain was intense—a heart-pang—lasting from 3 to 5 minutes. During the attacks he felt compelled to remain perfectly still. He was conscious of disturbed action of the heart during the attacks; the pain was confined to the præcordia. He was able to preach without difficulty, and stated that, were he exempt from the liability to these attacks, he should consider himself perfectly well. On examination of the chest, I found a feeble, soft systolic murmur at the base. The aortic valvular sound was loud. The heart was not enlarged. The intensity of the first sound precluded the supposition of fatty degeneration of the left ventricle.

This patient called upon me again in May, 1869, and reported much improvement. He relinquished his professional duties shortly after consulting me, and he had passed the winter quietly among his friends. The attacks of angina had ceased to recur, except on very active exercise, and the intensity of the pain in the attacks was much diminished. His general health was excellent. He was about to sail for Europe chiefly on account of the health of his wife.[1]

The diagnosis of angina pectoris is, in general, easily made, provided the practitioner have a clear idea of its distinctive features. Its paroxysmal character, the attack generally being remarkably abrupt, and often ending as abruptly; the brief duration, in most instances, of the paroxysms; the intensity of the neuralgic pain, and its radiation in different directions, in most cases extending to the left shoulder and arm; the sense of suffocation; the feeling of approaching death; the indescribable anguish; the pallor, anxiety, and apprehension depicted in the countenance—these are diagnostic characters which, in well-marked examples, leave no room for doubt as to the nature of the affection. Paroxysms of dyspnœa or so-called cardiac asthma, which are apt to occur in the progress of diseases of the heart, present points of difference so obvious that they need never be mistaken for paroxysms of angina. Their most prominent and distinctive feature, as the name dyspnœa implies, is difficulty of breathing. The sense of the want of breath is the chief source of suffering. The efforts of breathing are labored. The patient, instead of remaining perfectly quiet, is generally restless, frequently changing his position with the hope of finding relief. This difficulty of respiration does not belong to the history of angina. In the paroxysms of the latter the breathing is never extremely labored, and the patient often restrains voluntarily the respiratory movements, for fear of increasing his distress and sense of danger. Paroxysms of cardiac asthma rarely occur with the same abruptness as those of angina; nor do they end abruptly; and they are, as a rule, of much longer duration. They are rarely attended by acute pain. They are not characterized by that intense, undefinable anguish and feeling of approaching death which distinguish an attack of angina. A patient suffering from dyspnœa, however intense, looks forward to relief and comparative comfort. A patient attacked severely with angina feels that he is momentarily in danger of death, and that, were the paroxysm to continue, he must inevitably die. In short, if these two affections be sometimes confounded, it is from inattention to the points involved in the differential diagnosis.

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[1] He returned in November, 1869, and reported that his health was excellent.

It is somewhat less easy to discriminate between angina and neuralgic pains which may simulate, to some extent, paroxysms of the former. Instances, however, in which there is much real difficulty are very rare. Præcordial pain radiating perhaps in different directions, but not constituting angina, is most apt to be met with among the young, and oftener among females than among males. The acuteness of pain which characterizes attacks of angina, the anguish, and the sense of dying, are wanting. There is not the abruptness of the beginning and ending of the pain, nor the brief duration which characterize paroxysms of angina in the great majority of cases. The associated disorders are different. Persons affected with pseudo-angina are hysterical, anæmic, dyspeptic, &c., and the nervous system is manifestly disordered. On the other hand, persons are often attacked with angina when apparently in good health, and, at all events, the disorders just referred to are rarely found associated with it. The coexistence of organic disease of the heart is to be considered. In most instances of angina pectoris, cardiac lesions are determinable by physical signs, and this is a point entitled to weight in the diagnosis. In a large majority of the cases of angina we may expect to find, on exploring the chest, evidence of organic disease of the heart or of the aorta. If the pain, which bears some resemblance to that of angina, be due to intercostal neuralgia, the latter affection is determined by finding tenderness on pressure limited to circumscribed spaces, by the side of the spinous processes of the vertebræ behind, in the intercostal spaces on the lateral surface of the chest, and near the median line in front, tenderness at these points being diagnostic of that affection.

Neuralgic pain is sometimes incidental to the various forms of organic lesion of the heart, without constituting angina pectoris. As a symptom of cardiac lesions, exclusive of angina, it is rarely present in a marked degree, and is often wanting. Pain may be due to the coincidence of intercostal neuralgia, but in some instances it is evidently seated within the chest. Aside from other points, the absence of the strongly-marked paroxysmal character of angina suffices for the discrimination. There is no evidence that the existence of pain as an occasional symptom of organic disease of the heart, denotes greater liability to paroxysms of angina than if this symptom were not present.

The treatment of angina pectoris embraces, 1st. The means to be employed to diminish the severity and shorten the duration of the paroxysms; and, 2d. Measures in the intervals to postpone or prevent the recurrence of the paroxysms.

The severity of the pain during the attack, and its neuralgic character, point to the propriety of opium; and clinical experience shows that this remedy is more efficient than any other, in affording relief and bringing the paroxysm to a close. It is to be given in doses proportionate to the amount of suffering, and repeated after short intervals if the objects be not attained. Promptness of the effect of the opiate is highly important; and hence, the hypodermic mode of administration, whenever practicable, is to be preferred. If this mode be not practicable, the form of opiate selected to be given by the mouth should have reference to a prompt effect. Laudanum, the black drop, or an aqueous solution are preferable to the powder or solid gum, on account of the more speedy action of the former; but the salts of morphia are still more eligible in consequence of the ease with which they are given, and the greater certainty of their being retained. A convenient mode of administration is to place from half a grain to a grain upon the tongue. The succedanea of opium, such as belladonna, hyoscyamus, &c., are not sufficiently effective. They may be employed, however, when the paroxysms recur repeatedly during the day, and it is not deemed judicious to continue to prescribe opium freely. In proportion as the action of the heart is irregular and feeble, diffusible stimulants are indicated. They should be given after short intervals as long as they are indicated by these symptoms. Freely used they may, perhaps, be the means of averting sudden death. Brandy, or other kinds of spirit, may be employed; also the ethereal preparations and the carbonate of ammonia. Revulsive applications which act quickly, are indicated, viz., sinapisms, dry cupping, vesication with strong aqua ammonia, hot fomentations, and stimulating pediluvia. These several means are to be combined as convenience and the judgment of the practitioner may dictate in individual cases. The unexpected occurrence of the paroxysms and their brief duration, frequently render it impracticable to obtain medical aid before the attack has passed off. It is, therefore, important for the physician to give directions concerning the course to be pursued, in his absence, in the event of a recurrence of the paroxysms—an event to be expected, generally, sooner or later, after an attack has been once experienced. Efficient treatment may often accomplish much toward lessening the intensity, and perhaps the continuance of paroxysms in certain cases. But when the liability to their recurrence is such that they succeed each other after brief intervals, and are produced by slight causes, palliative measures, which unhappily are all that can be resorted to, succeed but imperfectly. I have known the inhalation of chloroform to be employed in a case of this description, the severest case that has come under my observation, with marked relief; and, indeed, this, after a time, was the only palliative that could be relied upon. The inhalation of ether, which may be resorted to with less apprehensions than chloroform, has been found by Carrière to be remarkably successful in arresting the paroxysms, and also in lengthening the intervals between their recurrence. If, as is possible, the inhalation of ether be unattended with danger, it is entitled to preference over other means of relieving the pain, in view of the greater promptness of its effect. Duchenne and Aran have found the electrization of the skin in the præcordial region, remarkably effective both in arresting the paroxysms and postponing their recurrence.[1] Swallowing pieces of ice has been found an effective measure by Romberg. It is to be added that, owing to the great variation in paroxysms as regards their duration, and the suddenness with which they often end, it is difficult always to determine whether they have been arrested by the measures employed, or whether they ended spontaneously.

The treatment having reference to the postponement or prevention of the paroxysms embraces the measures which are applicable to other neuralgic affections. Belladonna or atropia, quinia, arsenic, and zinc, are remedies which have been found useful and sometimes successful. Preparations of iron are indicated if the patient be anæmic, and they should be continued for a long period. The gouty and rheumatic cachexia claim appropriate treatment. Dyspeptic ailments and any other coexisting disorders are to be removed if possible. Habits of life which tend to impair the general health are to be reformed. Over-exertion of the mental faculties is to be avoided. These measures, if the affection be purely functional, will be likely to secure, in the end, permanent exemption from the paroxysms. They may be efficacious even if the affection be associated with cardiac or aortic lesions. In either case, if not completely successful, they may be useful in lengthening the intervals between the paroxysms, and lessening the severity of the latter.

In the cases in which lesions exist, these are to be determined as fully as practicable by means of symptoms and signs, and the treatment pursued which would be indicated if angina had not occurred. Judicious measures indicated by the coexisting lesions will be likely to prove useful, in a greater or less degree, with reference to the recurrence and the violence of the paroxysms of angina. At all events, the danger incident to the paroxysms may be diminished by these measures. Indications falling under this head have been already considered in treating of the different forms of organic disease of the heart in preceding chapters.

Exciting causes are to be avoided as far as practicable. Strong mental excitement, violent muscular exercise of any kind, and especially walking rapidly or ascending an acclivity against the wind, excesses in eating or drinking, &c., are apt to bring on an attack. By scrupulously avoiding these and other exciting causes which are peculiar to the individual experience of persons affected, it is highly probable that paroxysms may be warded off which would otherwise have occurred. Important, however, as is this part of the treatment, its effect is limited. Paroxysms often occur when they are not referable to any exciting cause. And when thus referable, it is probable that often the exciting cause (as the name implies) only determines the particular moment when the paroxysm takes place, anticipating somewhat the time when it would have occurred spontaneously.

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[1] *Vide* art. by Jaccoud, in Nouveau Dictionnaire, 1865.

ENLARGEMENT OF THE THYROID GLAND AND PROMINENCE OF THE EYES.

The occurrence of enlargement of the thyroid gland and prominence of the eyes, in connection with cardiac disorder, may be conveniently noticed in the present connection, although these events by no means necessarily involve the existence of any organic affection, and that they depend on an abnormal state of the heart cannot be considered as conclusively established. It is only within late years that the attention of clinical observers has been directed to these events, as connected with disturbance of the heart's action. The coincidence of enlargement of the thyroid gland with affections of the heart was observed by Parry in seven cases.[1] A few instances had previously been reported. Subsequently, Graves dwelt upon this coincidence in his *Lectures on Clinical Medicine*, published in 1835, giving some cases that had fallen under his observation. Afterward, the coexistence of prominence of the eyes, together with enlargement of the thyroid gland, attracted attention. Cases were reported by Stokes, McDonnell, and Marsh, of Dublin. Stokes, in his work on the *Diseases of the Heart and the Aorta*, devotes considerable space to the consideration of the subject. In our own country, Dr. Isaac E. Taylor contributed an elaborate paper, containing an account of two cases which he had observed.[2] Begbie, of Edinburgh, and Robert Taylor, of

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[1] "Collections from the unpublished medical writings of the late Caleb Hilliard Parry, M.D.," London, 1825.

[2] New York Medical Times, vol. ii, No. 3, December, 1852. Accounts of two cases, with remarks, and references to the literature of the subject, by Prof. H. J. Bigelow, Dr. Morland, and Dr. John S. Flint, are contained in an article in the Boston Medical and Surgical Journal, vol. lxi, No. 2, August 11th, 1859.

London, also reported cases, the latter giving the details of four cases which had fallen under his own observation, and an analysis, with regard to certain points, of twenty cases collected from various sources.[1]

The foregoing brief historical sketch was given in the first edition of this work (1859). Since that date, further contributions to the literature of the subject have appeared, of which may be mentioned a monograph, by Fischer, published in the *Archives de Médecine*, and a lecture by Trousseau, contained in his *Clinique Médicale*. The former of these publications embraces an account of twenty-four cases. The events which have been named, to wit: enlargement of the thyroid body, prominence of the eyes, and cardiac disorder, form a triad constituting the striking characteristics of an individual affection. The affection has received different names. Trousseau designated it "Graves's disease," in recognition of the fact of its having been first considered as an individual affection by Graves, of Dublin, in 1835. This name is often applied to it in this country. Graves, however, had not observed prominence of the eyes as an element of the affection prior to the communication of a case by Stokes. A German observer, Basedow, published an account of the combination of the three symptomatic events in 1840, and hence, in German medical literature, the name "Basedow's disease" is often used. Other names are: exophthalmic goitre, exophthalmic cachexia, and anæmic protrusion of the eyeballs. The latter name implies that an essential pathological condition is anæmia.

The affection is of rare occurrence. When the first edition of this work was published, I had met with only a single case in which the three symptomatic events were combined. I have recorded only five cases during the last ten years, in a wide field of clinical observation. I have notes, therefore of but six cases. As evidence of the infrequency of its occurrence, in a service at Bellevue Hospital during eight months of the year, for the past eight years, two cases only have come under my observation.

Of the triad of events, prominence of the eyes is the most striking and distinctive. Both eyes are affected, and generally in about an equal degree. The protuberance of the globes renders visible a broader portion of the tunica albuginea surrounding the cornea than is usual, and this gives to the face a wild, staring, ferocious expression which is remarkable and characteristic. In some cases the protrusion of the eyeballs is so great that the lids are unable to cover them, and the patient sleeps with the eyes partially open. Under these circumstances the conjunctivæ may become, after a time, inflamed, and ulceration of the cornea may take place, but, in general, there is no inflammation, and the eyes sometimes have an unusual brilliancy. They are sometimes irritable and watery. The pupil is not affected. There is no strabismus. The eyes are projected directly forward, that is, not inclining either in an upward or downward direction. Vision is unimpaired. There is no pain referable to the eyes, but there may be, for a time, an uncomfortable feeling as if the eyeballs were starting from their sockets.

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[1] London Medical Times and Gazette, May 24th, 1856, and the American Journal of Medical Sciences, July, 1856, p. 258.

The prominence of the eyes is not due to enlargement of the eyeballs, although generally the size of the latter is somewhat increased. As a rule, the eyeballs may, with gentle pressure, be returned to their natural situation within the orbits, the protrusion taking place instantly the pressure is suspended. It is probable that the primary cause of the exophthalmia is an enlargement of the vessels within the orbit, the morbid condition being the same as that which probably occasions the coexisting enlargement of the thyroid body. The protrusion, in some cases, may be increased by more or less serous effusion in the intra-orbital areolar tissue, and perhaps, also, as suggested by Dalrymple, by "an absence of the proper tonicity of the muscles, by which the eyes are retained in their natural positions in the orbit." Examinations after death have shown enlargement of the branches of the ophthalmic artery, a varicose condition of the veins within the orbits, serous effusion in a case in which anasarca had existed and disappeared, and hyperplasia of the intra-orbital areolar tissue. Broca, on the other hand, found no anatomical changes to account for the exophthalmia. As regards appearances ascertained by the ophthalmoscope, congestion and pigmentary deposits in the retina were observed by Withuisen, and no important changes by Follin.[1] Serous effusion and a morbid development of the areolar tissue within the orbit, together with the deposit of pigment in the retina, are, probably, secondary to, and dependent upon, congestion of the vessels; and it is intelligible that if the latter alone existed, the blood leaving the vessels after death, there may be no appearances found, on post-mortem examination, to account for the exophthalmia.

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[1] *Vide* Trousseau, Clinique Médicale.

The protrusion of the eyeballs sometimes takes place suddenly. In a case observed by Stokes, it was first noticed after a long-continued fit of coughing and retching. An instance in which it occurred during a single night is cited by Robert Taylor. In most cases it is at first slight, and it gradually increases until it reaches a certain degree, when it remains stationary. The latter statement is true of all the cases which I have observed, save one case. In this case the exophthalmia, which was slight, occurred suddenly after violent coughing.

The degree of the enlargement of the thyroid gland varies much in different cases. In two of my cases it was so slight as not to have attracted the attention of the patients. It is, in most cases, moderate in amount. Like the exophthalmia, it gradually increases to a certain degree, and then remains stationary for an indefinite period. Generally the enlargement is greater on one side, which may be either the left or the right side, oftener the latter. Exceptionally the enlargement is so great that respiration is obstructed by pressure on the trachea. The enlarged gland may also, by pressure on the recurrent laryngeal nerves, interfere with the movements of the glottis in respiration and speech. I have not met with a case in which the goitre was more than moderate in amount.

Pulsation of the thyroid arteries is a remarkable feature. A strong, expansive movement is felt by the hand, and is apparent to the eye. The carotid arteries pulsate strongly. Frequently there is a tactile thrill analogous to that of an aneurism. Graves and Vidal have each met with a case in which the affection was mistaken for aneurism. On applying the stethoscope, a loud bellows-murmur is perceived, which may be either systolic or continuous, the latter resembling the murmur of an aneurismal varix. Frequently, but not invariably, the venous hum is found on the neck without the space occupied by the goitre.

The thyroid enlargement is attributable, primarily and chiefly, to an increased size of the vessels; in other words, to an abnormal accumulation of blood, or a congestion, which is both arterial and venous. Examinations after death have shown that the arteries and veins become greatly enlarged ; also, the anatomical elements of the thyroid gland have been found hypertrophied together with an abnormal quantity of areolar tissue, and numerous small cysts enter into the appearances after death.

The third of the triad of events which characterize this affection, relates to the action of the heart. The heart acts with notable rapidity. The frequency of the pulse varies from 100 to 140 per minute. At times the frequency may even exceed considerably 140. Persistingly frequent, its frequency is greatly increased by exercise and mental excitement. When unusually increased, it may be irregular and intermitting, but, as a rule, the habitual disturbance is simply increased frequency. The force of the radial pulse is not abnormally great ; in this respect it is in striking contrast with the thyroid and carotid arteries. In some cases the abdominal aorta and cardiac arteries beat with abnormal force. The patient is generally conscious of the heart's action, and experiences more or less annoyance from it. The apex-beat gives a sensation of violence or shock, and often communicates to the præcordia a jarring movement. The sounds are sharp and loud, and the apex-beat is sometimes accompanied by tinnitus. In short, a persistent palpitation characterizes this affection, a palpitation continuing uninterruptedly, not only for many months, but, in some cases, for a great number of years.

I have included this affection among those incidental to diseases of the heart. At the date of the publication of the first edition of this work, it was a question whether or not, the affection is always associated with cardiac lesions. This is not a question at the present time. It is certain that the affection occurs independently of any organic disease of the heart. In the cases in which valvular lesions, with or without enlargement, coexist, the coincidence is accidental; but it may be that cardiac lesions tend to increase the disturbed action of the heart. It is, however, an interesting and important fact, that enlargement of the heart may be a result of its long-continued increased action. Of this fact, one of my cases afforded a striking illustration. In the case referred to, when the patient first came under my observation the heart was not enlarged; but after several years, the palpitation continuing, notable enlargement was found to have ensued. An account of this case will be presently introduced. The case is valuable as apparently demonstrating the occurrence of enlargement of the heart purely in consequence of long-persistent abnormal functional activity. In the affection under consideration, then, the element which concerns the heart is primarily a purely functional disorder; and, in the arrangement of topics, it would have been more correct to consider the affection as incidental to the latter rather than to organic diseases of the heart. Of the six cases which I have observed, in one case only was there evidence of valvular lesions and enlargement. In all the other cases, when they first came under my observation, there was no enlargement of the heart. In one case, as just stated, it was known that enlargement subsequently took place. In one of the cases the affection had existed for five years without having led to enlargement.

As regards the order of the occurrence of the three events which characterize this affection, the cardiac precedes the others. Increased action of the heart exists for weeks or months prior to either enlargement of the thyroid gland or prominence of the eyes. This rule is invariable. Of the two other events, either may take precedence in following the palpitation; the exophthalmia, in some cases precedes the goitre, and in some cases, the latter first occurs. Moreover, of the three events, the functional disorder of the heart only is never wanting. Cases occur in which prominence of the eyes and palpitation are not accompanied by any enlargement of the thyroid gland. In two of my cases the latter was so slight as to have escaped the patients' notice. Cases, on the other hand, of enlargement of the thyroid gland with palpitation, and without any prominence of the eyes, are less infrequent. In the first edition of this work I introduced an account of two cases of description which had fallen under my observation. These cases were under my observation for a short time only, and prominence of the eyes may have subsequently taken place. Undoubtedly cases in which either enlargement of the thyroid gland or prominence of the eyes is associated with the cardiac element, are to be regarded as cases of the affection under consideration. Prominence of the eyes did not exist in the affection as first described by Graves.

Aside from the three events which have been considered, other morbid conditions enter into the clinical history. Anæmia is generally present, and is often a notable feature, as shown by pallor of the prolabia and face, a blanched appearance of the mucous membrane of the mouth, and by blueness of the eyeballs. These visible characters of anæmia, however, are present in only a certain proportion of cases. When they are not present, the existence of anæmia may be shown by the venous hum in the neck and by inorganic murmurs at the base of the heart; but these evidences of anæmia are sometimes wanting. Although a frequent concomitant, and frequently existing in a marked degree, the anæmic condition cannot be said to form a constant element of the affection. Hence, it cannot, as some have supposed, have an essential pathological connection with either of the three events which characterize the affection. Disorders of digestion are not uncommon; but patients not infrequently eat sufficiently, digest without trouble, and the nutrition of the body is well maintained. Amenorrhœa is a frequent morbid condition. Dyspnœa on exercise is common, and is dependent chiefly on the unusual excitation of the heart which exercise occasions. Mental irritability, nervousness, and depression of spirits are prominent symptoms in most cases. They are attributable in part to anæmia, but perhaps more to the moral influence of the disease, especially if the exophthalmia be so great as to render the patient an object of curiosity and wonder. Insomnia is, in some cases, a distressing symptom. The affection is not incompatible with apparently excellent health in all other respects. In one of my cases the patient, a quadroon girl, aged 19, made no complaint of any ailment, although the action of the heart was very rapid, and the prominence of the eyes was marked. She was able to take active exercise without difficulty, and all the vital functions appeared to be well performed. The affection in this case had existed for a year.

Of the pathology of the affection no satisfactory exposition can be given. The primary and essential manifestations pertain to the heart, and consist in a hyperactivity of this organ. The antecedent, underlying pathological conditions are not known. As stated already, the characteristic events are not attributable to anæmia. Anæmia does not invariably exist, and, when present, it bears no fixed relation to the amount of palpitation. This, however, is not saying that anæmia may not often contribute, more or less, to the cardiac disorder. That it does so is highly probable, and, hence, in proportion as it enters into the affection, as a concomitant, it is, in a practical view, important to be considered. That a causative relation does not exist between the thyroid enlargement and the prominence of the eyes, is shown by the fact that sometimes one and sometimes the other occurs first, and also by the fact that each may be wanting. These events are always consecutive to the cardiac disorder, but whether they are dependent upon the latter, or whether they are effects of the unknown pathological conditions which determine the disordered action of the heart, our present knowledge does not enable us to decide.

Our knowledge of the causation hardly extends beyond the agency of sex and age. In the great majority of cases the patients are females. Of 50 cases collected by Withuisen, in 8 only were the patients of the male sex.[1] In all the cases which I have observed the patients were females. As regards age, the affection very rarely occurs either under puberty or after the middle period of life. In two of my cases the affection occurred shortly after severe mental trouble. Anæmia may, in some cases, contribute to its production; but anæmia, in some of the cases in which it coexists, is developed consecutively. It is needless, to state that anæmia is sufficiently common without being connected with this affection. In short, exclusive of age and sex, nothing positive is known of the etiology.

The diagnosis of the affection involves no difficulty. If exophthalmia be present in a marked degree, this alone is distinctive. The prominence of both eyes directly forward, without strabismus, together with the absence of pain, the preservation of vision, and, in general, the practicability of returning them to their normal position by pressure, are points sufficient to distinguish this affection from protrusion caused by certain diseases of the eyeball or tumors within the orbit. Protrusion from these causes is generally limited to one eye, and is rarely in a direct line without strabismus. The association of enlargement of the thyroid gland and of rapid action of the heart, the latter not dependent on cardiac lesions, renders the diagnosis of the affection positive. The association of persistent palpitation with the characteristic prominence of the eyes, without goitre, and, on the other hand the association of the persistent palpitation with goitre, and without prominence of the eyes, are sufficient for the diagnosis. As already stated, the affection is to be considered as existing when the prominence of the eyes, or the goitre, either without the other, is associated with the characteristic cardiac disorder. In the cases in which enlargement of the thyroid gland exists without prominence of the eyes, the affection is distinguished from ordinary bronchocele by, in general, the limited amount of enlargement, by the strong arterial pulsation together with the murmurs on auscultation—these being differential points, in addition to the persistent palpitation. The latter does not accompany ordinary bronchocele.

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[1] *Vide* Nouveau Dictionnaire, art. Cœur.

The prognosis, as far as regards a fatal result from the affection *per se*, is favorable. The affection does not tend intrinsically to destroy life. It is, however, an affection which continues indefinitely. It is a chronic affection, and it must impair, to a greater or less extent, the ability of the system to support and recover from other affections. Death generally occurs from intercurrent or superadded diseases. But without the occurrence of these, the affection ultimately leads to great feebleness and emaciation. On the other hand, it is sometimes well tolerated for many years.

In a certain proportion of cases the progress of the affection is favorable. The enlarged thyroid gland may gradually decrease in size, and at length the goitre may entirely disappear. The prominence of the eyes may gradually become less, but very rarely, if ever, do the eyes resume their normal appearance; more or less exophthalmia continues permanently. The palpitation may become less and less marked, and, if recovery take place, the rapid action of the heart ceases. Data are wanting to determine the proportion of cases which end in recovery, but it is certain that they are exceptions to the rule. As a rule, even in the cases in which a notable improvement takes place, complete recovery is not to be expected.

Enlargement of the heart induced by the long-persistent palpitation may hasten and determine a fatal termination. Exclusive of enlargement of the heart, the affection does not appear to involve a liability to disease of any of the organs of the body. The intercurrent affections which have been observed have not sufficient uniformity in different cases, and are not frequent enough, to show any special relation of dependence.

As bearing upon the prognosis, with reference to which especially the accumulation of recorded cases is desirable, I shall introduce in this connection abstracts of the histories of three of the cases which I have observed. In the remainder of the cases which I have noted, the patients were only temporarily under my observation.

Case 1. This case came under my observation in April, 1859. The patient was a girl, aged 16. She presented a healthful appearance, having good color. She had not had rheumatism, or any acute affection within the chest. She had been conscious of a rapid action of the heart for the space of a year. She was then at a boarding-school, and was seen by a physician who was much concerned at finding her pulse habitually from 120 to 130 per minute. For a time she was placed on a restricted diet, under which the heart's action became still more rapid. Prominence of the eyes had existed for five months. Enlargement of the thyroid gland had not been noticed, but the patient had felt a strong pulsation over it, especially on the right side.

When I first saw her the pulse was rapid, but its frequency is not noted. The eyes were prominent, but not to an extent to produce a very striking appearance. The thyroid gland was distinctly enlarged, the right lobe being more so than the left. A soft, systolic murmur was heard over each lobe, and a loud venous hum, together with a systolic carotid murmur, was found on the neck without the situation of the thyroid gland. The heart was not enlarged. There was no cardiac murmur. The heart-sounds were abnormally intense. The appetite and digestion were good. There was no disorder of the menses.

The measures of treatment employed in this case were, a generous diet, recreation, living as much as possible in the open air, and the internal use of belladonna.

Six years afterward, namely, in June, 1865, this patient made a friendly call upon me. She was then just married, and about to embark for Europe. Prominence of the eyes, especially of the right eye, was observable, but less than when she was formerly under my observation. There was still some enlargement of the thyroid gland, and the enlargement varied at different times. The heart was irritable, becoming rapid on slight exercise or excitement. Aside from the liability to palpitation, she stated that her health was good. I did not make a physical examination of the heart.

Three years after the foregoing date, namely, in September, 1869, being nine years from the date of her first consulting me, I received a communication from her father, in Switzerland, stating that she had been recently confined, the child dying a few days after birth from some difficulty connected with respiration; that she was very dropsical, and that her physicians said she had disease of the heart. Her death occurred a few days afterward.

It is probable that in this case the affection had led to enlargement of the heart.

Case 2. Bridget Sherwood, aged 28, seamstress, unmarried, was admitted into Bellevue Hospital, May 3d, 1861. She had been ill for five years, having previously had good health. Her illness began with intermittent fever, and during six weeks she had repeated relapses of this disease. At the end of this six weeks she noticed a swelling of the neck on the right side. This swelling progressively increased until it reached the size which it had on her admission. Swelling on the left side did not begin until four months before her admission. The swellings were in the situation of the thyroid gland. On her admission they were considerable, the enlargement of the right lobe of the thyroid gland being greater than that of the left. They were soft to the touch, and pulsated strongly. They had never been painful. About six months after the enlargement of the thyroid body on the right side was first noticed, it was observed that her eyes were prominent. The prominence increased, and, at the time of her admission, it existed in a marked degree, giving to the face a peculiar and characteristic appearance. She had had no pain in the eyes, and vision was unaffected. She was able to close the eyelids completely. She had been unable to work for six months, on account of debility, shortness of breath on exercise, præcordial pain, and palpitation. The appetite had been fair and the bowels regular. The menses had not appeared for the preceding six months, having previously been regular. She had suffered from disturbed sleep, and her spirits had been depressed. Her weight had considerably diminished.

Her aspect on admission denoted anæmia, but not in a marked degree. The pulse was habitually 128 per minute. It was small and weak at the wrist, presenting, in these respects, a striking contrast with the pulsations in the neck. The apex-beat of the heart was in the fifth intercostal space, and the dulness on percussion in the superficial and the deep cardiac space was not increased, either in degree or in extent. A systolic shock was felt in the præcordia, but no heaving. At the apex the heart-sounds were distinct, sharp, and loud, but without murmur. At the base, on the right of the sternum, there was a faint, low systolic murmur. A soft, rather high, and feeble murmur was heard over the body of the heart. An intense, continuous murmur was heard over the thyroid swellings. There was also tactile thrill.

She was treated with chalybeates, quinia, and, for a short time, with the veratrum viride. The latter was soon suspended on account of unpleasant effects.

December 22d, 1861. It is noted that the patient's condition was not improved. At this time the patient was not under my care.

February 3d, 1862. The patient came again under my care. She reported improvement, and her appearance was evidently better than at the time of her admission. The anæmia was less; the thyroid swellings had decreased; she had less palpitation, and she was stronger. Her appetite was now excellent. She had taken the citrate of iron steadily for three months, and it was continued.

March 20th, 1862. This patient was discharged at her request. She had progressively improved. The enlargement of the left lobe of the thyroid gland was nearly gone, and on the right side it was slight. The eyes were less prominent. Her aspect, as regards anæmia, was better. The action of the heart had notably diminished.

This patient was readmitted March 29th, 1863, but not in my division. I have noted under this date that she had worked for ten months in an establishment for preserving meats and fruit, and that she stated her health to have been "first-rate." In the early part of the month (March, 1863), she took cold as she thought; the breathing became hurried, and the palpitation returned. On her readmission, the anæmic appearance was slight; the prominence of the eyes was much less than formerly; the left lobe of the thyroid gland was slightly, and the right lobe moderately, enlarged. The action of the heart was rapid. The apex-beat was in the fifth intercostal space on the linea mammalis, and the superficial cardiac space was somewhat enlarged. There was an aortic direct murmur, and no other cardiac murmur was discernible. On the neck, on both sides, there was a loud, continuous murmur which was not suspended by pressure upon the veins above the point of auscultation.

April 28th, 1868. I found this patient in Charity Hospital, Blackwell's Island. She had been in this hospital for twelve months. She was not in my division. She was emaciated and feeble. The appetite and digestion were tolerably good. The prominence of the eyes was slight, and there was very little enlargement of the thyroid gland. I noted, in addition, under the above date, as follows: "An interesting fact in this case, is the development of hypertrophy of the heart. The apex-beat is now in the sixth intercostal space, at least two inches without the linea mammalis, and the area of præcordial dulness, and also its degree, are notably increased. There is no cardiac murmur. I can discover no other disease than that of the heart."

On entering upon duty at Charity Hospital, March, 1869, I found this patient still there. She was quite feeble, keeping, for the most part, the bed. She was also much emaciated. The pulse was still rapid. The prominence of the eyes was slight, and the thyroid gland was not much enlarged. The thrill and murmur over the thyroid bodies had gone. She was subject to diarrhœa. The enlargement of the heart was considerable, without any cardiac murmur.[1]

CASE 3. Miss B., aged 29, was referred to me by my colleague, Prof. Van Buren, March 6th, 1866. The eyes were notably prominent, but they had been even more so. She was unable to cover the globes with the lids. There was no defect of vision except myopia. The thyroid gland had been enlarged, but it was not so at this time. The action of the heart was very rapid, the pulse being 140. She was always conscious of excessive action of the heart, and at times the palpitation was distressing. There was no cardiac murmur, and the heart was not enlarged. She had not an anæmic aspect, but there was a loud venous hum in the neck. Her general health was good. She had of late increased in weight. The appetite and digestion were excellent. The menses were regular but scanty. The affection had existed for five years. Prominence of the eyes occurred prior to the thyroid enlargement, but palpitation preceded both. She had been treated by Prof. Van Buren for several years, and, for a time, by Prof. Hammond. Iron, digitalis, and electricity had been employed. I saw her repeatedly during several months from the date of her first consulting me, and made trial of the bromide of potassium with no apparent benefit. I have not seen her for more than two years; but I am informed that her condition at the present time (August, 1869), is better than when she was under my observation.

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[1] This patient died in August, 1869. The record of the autopsy states that the heart weighed 20 ounces. No mention is made of any disease of the valves. The lungs were emphysematous. There was no tuberculous affections. The liver weighed 30 ounces, and appeared healthy. The spleen weighed 8 ounces. The kidneys weighed 14½ ounces, and were very firm—fibrous. The thyroid bodies were of the size of two eggs. The record book contains no account of the appearances within the orbit. The ante-mortem history after March, 1869, was not noted, and the case was not under my observation after that date. The condition of the kidneys suggests the question whether the enlargement of the heart may not have resulted from renal disease.

This case is introduced chiefly in illustration of the persistence and the tolerance of the affection for a long period (eight years), the absence of any serious complications, and the ground of a favorable prognosis as regards further tolerance of the affection, if not progressive improvement and ultimate recovery.

The *rational* objects of treatment, in cases of this affection, relate to the palpitation, the excitability of the nervous system, the existence of anæmia in a large proportion of cases, and to the functions subservient to nutrition. With reference to the palpitation, digitalis and the veratrum viride, employed in some of my cases, have not appeared to be of use. The long-continued use of aconite deserves to be tried. As a nervous sedative, belladonna in one of my cases seemed to be useful. Chalybeate remedies are indicated in proportion as anæmia is marked. These remedies should be continued for a long period. Other tonics are indicated if the appetite be impaired and digestion difficult. Phosphorus, zinc, and arsenic, are remedies which may be suggested as worthy of trial.

The hygienic treatment is highly important. An abundant alimentation is indicated. Care should be taken to avoid unusual disturbance of the heart by active exercise or mental excitement. Healthful recreation is to be secured as far as practicable. The hydropathic treatment is advocated by Trousseau, who cites cases in which it proved highly effective. It should be employed tentatively, and continued if it prove beneficial. In the regulation of diet, while nutritious articles should be advised, and in ample quantity, those which stimulate the stomach and thereby excite the heart are to be avoided. The inquiry arises, whether a purely milk and farinaceous diet might not in some cases conduce to improvement.

REDUPLICATION OF THE HEART-SOUNDS.

Reduplication, or doubling of one or both of the sounds of the heart, is an auscultatory sign which was not considered in treating of organic affections, because it has not been found to be incidental to any particular form of cardiac lesion, and it occurs independently of organic disease. Although, as a physical sign, it has not much practical value, it claims attention as denoting an interesting aberration of the heart's action, and it is important for the practitioner to be prepared to recognize it, in order that he may appreciate what would otherwise be an unintelligible anomaly. Either or both of the sounds may be reduplicated. Reduplication of the second or diastolic sound, however, is the variety generally observed. This is not uncommon. Instances in which the first sound is alone doubled, and of the reduplication of both sounds are so rare that they may be included among the curiosities of clinical experience. Using for the present purpose the expression *tic-tac* as the symbol representing the normal sounds, the three varieties of reduplication may be verbally expressed thus: Doubling of the first or systolic sound, by tic tic-tac; of the second or diastolic sound, by tic-tac tac; of both sounds, by tic tic-tac tac. In the two first varieties, or when one only of the sounds is doubled, three sounds occur during a single beat or revolution of the heart; in the last variety, or when both sounds are reduplicated, four sounds are heard with each revolution or beat. Bouillaud compares the rhythmical succession of the triple sounds, when the second sound is doubled, to the dactyle in poetical metre, and the rebound of a hammer on an anvil; and when the first sound is doubled, to the tattoo (rappel) of the drum, or the sounds of the feet of a galloping horse.

Bouillaud claims to have been the first to describe reduplicated heart-sounds. He states that, in the first edition of his treatise on *Diseases of the Heart*, published in 1834, he was able to cite but a single instance; but, before the appearance of the second edition in 1841, he had collected several examples, so that the occurrence of reduplication could no longer be discredited, as it has been by some.[1] The reality of the sign has been now abundantly confirmed, and, as just stated, examples of the reduplication of the second sound are not uncommon.

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[1] **Leçons Cliniques sur les Maladies du Cœur, &c., par M. Bouillaud, recueillies et rédigées par le Dr. V. Racle, Paris, 1853.**

Bouillaud considered reduplication of the heart-sounds as always incidental to organic diseases of the heart. This was the opinion generally held when the first edition of this work was published. Undoubtedly the sounds are reduplicated oftener with, than without, cardiac lesions; but it is certain that the latter are not essential. I shall presently introduce a case in which a post-mortem examination showed the heart to be free from any morbid appearances. It occurs to me not infrequently to meet with reduplication of the second sound when there are no physical evidences of organic disease. A French observer, Potain, has found that transient reduplications are discoverable in a considerable proportion of persons in health, if attention be directed to the sounds at particular periods of respiration. He states that a normal reduplication of the first sound is liable to be heard at the end of the act of expiration, and the beginning of the act of inspiration. He attributes its production to the pressure of the blood in the right side of the heart causing the closure of the tricuspid valves to lag a little behind the closure of the mitral valves. On the other hand, a normal reduplication of the second sound is heard at the end of inspiration and the beginning of expiration, the pressure of the blood within the aorta causing the closure of the aortic valves a little before the closure of the pulmonic valves.[1] Morbid reduplications are not thus influenced by, but occur independently of, the acts of respiration.

As regards the mechanism of reduplication of the heart-sounds, the explanation at first offered by Bouillaud was, that the systolic and diastolic movements of the ventricles take place synchronously, but consist each of two distinct efforts (*reprises*); that is to say, each systole and diastole is divided into two acts, and each act attended by a sound. The disorder of rhythm, according to this hypothesis, is analogous to interrupted or jerking respiration. It is assumed in this explanation, that the diastolic sound of the heart is due to an active dilatation of the ventricles, and not to the recoil of the aorta and pulmonic artery. In his *Leçons Cliniques*, published in 1853, however, Bouillaud adopts the explanation now generally received, as more satisfactory than that previously offered by himself, namely, that the reduplication is caused by the failure of the two ventricles to contract in unison. According to this explanation, the tricuspid and mitral valves are made tense, not simultaneously, but successively, in consequence of the contraction of one ventricle being completed before that of the other; and the semilunar valves of the aorta and pulmonic artery expand alternately, instead of coincidently, since, in consequence of the difference in time between the completion of the contractions of the right and left ventricle, the recoil of the coats of each of these two vessels does not occur at the same instant. Hence, the conditions for the doubling of both sounds always exist, although one sound only, either the first or second, may be reduplicated; but it is readily conceivable that one only may be reduplicated with intensity sufficient to be appreciable by auscultation. The fact, also, that the reduplication of the first sound is appreciable very rarely in comparison with the reduplication of the second sound, is susceptible of explanation. The valvular element of the first sound is weaker, as a rule, than the second sound of the heart. When the element of impulsion is eliminated from the first sound, the second sound becomes accentuated; that is, its intensity is greater than that of the first sound. Now, in general, when the heart-sounds are reduplicated, the action of the organ is weakened, and the element of impulsion of the first sound is either lessened or wanting. Under these circumstances, the tension of the tricuspid valves will not be likely to be appreciable on auscultation; hence, reduplication of the first sound, although it actually occurs, is not discoverable. As rare exceptions to the rule, the tension of the tricuspid and the mitral valves may each be with such force that the reduplication of the first sound is apparent, although the reduplication of the second sound fails to reach the ear. And, again, the sound from each of the four valves may be loud enough to be heard, and then the heart-sounds are quadrupled.

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[1] *Vide* Art. Cœur, by Luton, in Nouveau Dictionnaire, 1868, tome viii, p. 801.

Walshe remarks, that "the real interest of reduplications arises out of their bearing on the theory of the heart-sounds." He asks: "How is the fact that the second sound may be continuously doubled at the base, and perfectly pure and single at the apex, explicable on the simple sigmoid theory of the second sound?" He adds: "A double sound does not become single by conduction over so short a space." This fact seems to me to be susceptible of an easy explanation, without conflicting with the theory which refers the production of the second sound to the semilunar valves. Examinations of the healthy chest show that the pulmonic second sound is weak, as compared with the aortic second sound. The former is only distinguishable, as a rule, in the first, second, or third intercostal spaces on the left of the sternum. The second sound heard over the apex and elsewhere, more or less removed from the points just named, emanates from the aorta. Hence, when the second sound is reduplicated at the base of the heart, the reduplication may not extend to the apex, simply because the pulmonic sound is not propagated so far. Owing to the relative weakness of the pulmonic second sound, the reduplication may be appreciable at the base of the heart on the left, and not on the right side of the sternum. Again, the fact that "the first sound may be single at the left apex and at the base, while it is distinctly reduplicate at the right apex," appears to Dr. Walshe to denote that the first sound consists of a ventricular and an arterial portion, and that the two are separated on the right side of the heart. But this fact seems to be explicable readily by the weakness of the tricuspid valvular element of the first sound, as compared with the mitral valvular element. The tricuspid valvular element is appreciable only over the right ventricle; hence, a doubled first sound may be heard in that situation only, simply because the tricuspid sound is not strong enough to be transmitted to the left apex and to the base. Finally, Walshe cites the fact that the second sound may be single at the base and double at the left apex, as tending to show strongly the partial origin of the second sound within the ventricle. This fact is not so readily explained, and I cannot but think that, whenever the second sound is doubled at the left apex, it will be found to be reduplicated over the pulmonic, although it may be single over the aortic, artery. The pulmonic sound may, in some instances, be propagated as far as the apex of the heart, especially when the right ventricle is hypertrophied, while it is not appreciable over the aorta.[1]

Reduplicated heart-sounds are distinguished, in general, without difficulty, if the auscultator be prepared to recognize them by a proper knowledge of the subject. The occurrence of three sounds with a single beat is easily determined, and the rhythm shows, at once, whether it be the first or the second sound which is doubled. The latter, it is to be borne in mind, is reduplicated much oftener than the former, and this variety of reduplication, as a rule, is most marked, and it may be perceived only at the base of the heart. As just stated, it will be most likely to be discovered over the pulmonic artery, that is, in the second intercostal space on the left side of the sternum. Reduplication of the first sound is most likely to be observed over the right ventricle. The movements of the apex of the heart against the walls of the chest, in some cases, give rise to a prolonged and interrupted sound (element of impulsion) which may be mistaken for reduplication of the first sound. True reduplication of this sound is irrespective of the element of impulsion, being due to the disconnection of the tricuspid and mitral portions which together form the valvular element of this sound. Difficulty of diagnosis pertains chiefly to cases in which both sounds are doubled. In these cases the sounds may succeed each other so rapidly that the systolic and diastolic are hardly distinguishable by means of rhythm. The fact of reduplication in such cases is to be determined by comparison of the number of sounds with the apex-beat, if perceptible, or with the carotid pulse.

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[1] In connection with these remarks, the reader is referred to the analytical decomposition of the two sounds of the heart into, 1st, an aortic and a pulmonic valvular element composing the second sound; 2d, and a tricuspid valvular element, a mitral valvular element, and an element of impulsion, composing the first sound. *Vide* Chapter I, page 63.

There is a liability to error, if the comparison be made with the radial pulse; and a case introduced in the first edition of this work, as a case in which both sounds of the heart were reduplicated, is an illustration of the error now referred to. This case, and two other cases, I shall now give as examples of a variety of functional aberration of the heart's action, which, without due attention, may be confounded with reduplication of both heart-sounds.

Case 1. The patient was a seaman, aged 27, admitted into the Louisville Marine Hospital, for a cough which he attributed to taking cold six weeks before his admission. On comparing the pulse of the wrist with the heart-sounds, there were four sounds for each pulse; the number of double sounds was precisely twice as many as the number of radial pulsations per minute. This exact ratio was invariably preserved whenever the comparison was made during seventeen days. The carotid pulse, however, occurred in the ratio of one to every two sounds of the heart; in other words, it was precisely twice as frequent as the radial pulse. The heart was somewhat enlarged, and a feeble, short, systolic murmur was heard at the apex. The apex-beat was not appreciable to either the eye or touch. The patient was walking about, and was able to take active muscular exercise. Subsequently, the face and lower limbs became œdematous, and he suffered from dyspnœa. These symptoms disappeared, and he was discharged, apparently quite well, after having been two months in hospital. When discharged, the radial pulse and the sounds of the heart were in normal ratio, and the murmur had disappeared.

This patient remained, apparently, in perfect health, performing active labor for five years afterward. He then died with some acute affection, and an examination of the heart by my friend, Prof. T. G. Richardson, showed moderate enlargement without valvular disease.

This case I formerly considered as exemplifying reduplication of both heart-sounds; but there was a difficulty in reconciling with the fact that the ratio of the carotid pulse with the heart-sounds was normal, the explanation which has been given of reduplication, namely, want of synchronism of the action of the two ventricles. To meet this difficuly, it was supposed that the frequency of the carotid pulse was attributable to dicrotism. A more satisfactory view of the case, however, is that it was not an example of reduplication of the heart-sounds, but a case in which every alternate systole of the left ventricle was not strong enough to produce an appreciable radial pulse. That each alternate systole should be uniformly and regularly thus weak during seventeen days, is a very curious fact; but, perhaps not more curious than that want of synchronism in the contraction of the ventricles should continue steadily for that length of time. Another curious fact in this case is, assuming the explanation now given to be the correct one, the number of ventricular systoles per minute were 160, this being the frequency of the carotid pulse, the radial pulse being 80 per minute.

Case 2. A male patient, aged 20, was admitted into Bellevue Hospital, January, 1861, with general paralysis. On the day after his admission, I found that the two heart-sounds occurred twice for each radial pulse. The rhythm of the heart-sounds was normal, but in every alternate occurrence they were notably feeble. The radial pulse numbered 36 per minute. There was no cardiac murmur, and the heart was not enlarged. On the next day the same discrepancy existed between the heart-sounds and the radial pulse. On the next day but one reduplication of the second sound of the heart was observed for a few moments, when it ceased, and the discrepancy just stated did not exist. Subsequently, the second sound was repeatedly reduplicated. Death occurred suddenly ten days after his admission. On examination after death, the heart weighed ten ounces, and was in all respects healthy. The valves were sound, and the walls presented the appearance of healthy muscular structure. The carotid pulse is not noted in the history of this case.

CASE 3. Mr. C., aged about 45, was seen by me in consultation with Prof. Foster Swift, July 23d, 1868. The heart was moderately enlarged, and there was a short, soft systolic murmur over the apex. There were two sets of heart-sounds in this case for each radial pulse. The first and second sound, which were in normal relation with the radial pulse, were moderately loud. They were succeeded by a first and a second sound which were quite feeble, appearing to be an echo of the former, and without any radial pulse. At first, I supposed that the first two sounds might possibly be due to the systole of the left ventricle, and that the two sounds which followed were due to the systole of the right ventricle; in other words, that the two ventricles acted separately, the sounds from the auricular and the semilunar valves on one side taking place before the occurrence of the corresponding sounds on the other side. But, on further examination on the following day, this supposition was abandoned. The two sets of sounds existed, as on the day previous, one set being weak and the other set comparatively strong; but sometimes a feeble, radial pulse was felt with the weak sounds, and, if not, the radial pulse was proportionately infrequent; with both sets of sounds it was 100 per minute. Moreover, the carotid pulse was felt with each set of sounds. The patient suffered much from dyspnœa and vertigo. He passed, soon after my visits, from under the observation of Dr. Swift, and his death occurred not long afterward.[1]

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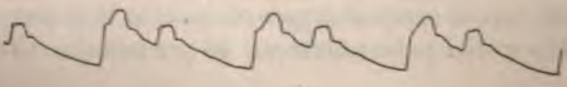
[1] Marey has represented this variety of functional aberration, as delineated by the sphygmograph, in the following figure, which gives, in a regular series, two consecutive pulsations, one strong and the other feeble.



Reduplication of the heart-sounds, like the aberration illustrated by the three cases just given, is one of the varieties of functional disorder of the heart. It occurs in cases of organic disease of the heart, but not exclusively in connection with any particular lesions, and it occurs when the heart is free from any structural affection. In its pathological import and diagnostic significance, it is a sign of very little value. Bouillaud calls it *une signe de luxe*, from its superfluousness in diagnosis. Generally, it is transient, occurring at irregular intervals, and varying in duration. The sounds may be tripled at one time, and quadrupled at another time, a fact readily explained by the difference in the intensity of the heart's action at different periods. Occurring in connection with organic disease, it does not render the prognosis more unfavorable; and occurring without any lesions of structure, it betokens no danger, nor does it give rise to any serious inconvenience. In short, with our present knowledge, the interest belonging to it, if the antithesis be allowable, is scientific rather than practical.

The treatment, in the cases in which reduplication occurs, will have reference entirely either to coexisting cardiac lesions, or to the morbid conditions with which it is associated. In this point of view, therefore, the subject does not claim consideration.

Page 420, Marey's work, Marey states that, on auscultation, the sounds of the heart appeared to be reduplicated. Both pulsations, in this case, were felt at the wrist, and the sensation was that of a dicrotic pulse. Assuming that the feeble pulse was due to dicrotism, the number per minute would have been 35; but, reckoning the feeble pulsations as due to the ventricular systole, the number was 70.

CHAPTER VII.

INFLAMMATORY AFFECTIONS OF THE HEART—PERICARDITIS.

Acute pericarditis—Anatomical characters—Division into three stages or periods—White spots on the heart—Pathological relations and causation of pericarditis—Connection with acute rheumatism, with albuminuria or Bright's diseases, with endocarditis, &c. —Symptoms of acute pericarditis—Symptoms referable directly to the heart, to the circulation, to the respiratory system, to the digestive system, to the countenance, position, &c., to the nervous system—Notable disorder of the brain and spinal cord in connection with pericarditis—Physical signs of acute pericarditis—Signs furnished by percussion, auscultation, palpation, inspection, and mensuration—Summary of the physical signs of acute pericarditis—Diagnosis of acute pericarditis—Prognosis in acute pericarditis—Treatment of acute pericarditis—Bloodletting, mercurialization, sedatives, revulsives or counter-irritants, opium, stimulants, and eliminatives—Treatment prior to liquid effusion, during the period of liquid effusion, and after absorption of liquid effusion—Treatment when complicated with notable disorder of the nervous system—Subacute and chronic pericarditis, with and without liquid effusion—Symptoms, physical signs, and treatment—Paracentesis of the pericardium—Pneumo-pericardium and pneumo-pericarditis—Pericardial adhesions—Effects upon the heart and circulation—Diagnosis.

INFLAMMATION affecting the heart may be limited to either of the anatomical structures which compose this organ. The investing serous membrane may be alone inflamed, constituting the affection called *pericarditis*. When the membrane lining the cavities, or the endocardium, is the seat of inflammation, the affection is called *endocarditis*. Inflammation of the substance or muscular tissue of the organ is distinguished as *carditis* or *myocarditis*. Although these different inflammatory affections may exist, each independently of the others, they are often associated. In a large proportion of the cases of pericarditis, endocarditis coexists; and myocarditis very rarely occurs save in connection with inflammation of either the investing or lining membrane of the heart. The intrinsic importance of these affections renders their study highly important. They are seated in an organ entitled to be called, *par excellence*, a vital organ. They involve, not infrequently, great suffering and imminent danger to life.

They derive importance from their remote consequences. The organic affections which have been considered in previous chapters, originate, in the majority of instances, in cardiac inflammation. The study of this class of affections has been rendered highly interesting and important by the developments of modern researches as regards their pathological relations, especially to rheumatism and renal disease, and by the improvements in diagnosis arising from the successful application of physical methods of examination.

PERICARDITIS.

Inflammation affecting the investing membrane of the heart, or pericarditis, is less frequent in its occurrence than endocarditis, but it is a more serious affection as regards immediate danger. This membrane is analogous in structure to serous tissue in other situations; and pericarditis does not differ essentially from pleuritis or peritonitis. The points of difference pertaining to the symptomatic phenomena and the dangers peculiar to it depend on the comparatively small size of the pericardial sac, the fact that the substance of the heart consists of muscular tissue, the function of the organ and its physiological relations. In treating of pericarditis, the morbid changes incident to the disease, or its anatomical characters, will be first considered; and next, its pathological relations and causation. The symptoms, physical signs, diagnosis, prognosis, and treatment, severally claim distinct consideration. This, like other inflammatory affections, is presented in an acute, a subacute, and a chronic form.

I shall treat, in the first place, of acute pericarditis under the foregoing heads, treating afterward of subacute and chronic pericarditis; and, finally, pericardial adhesions will claim some attention.

ANATOMICAL CHARACTERS OF ACUTE PERICARDITIS.

The morbid changes found after death in fatal cases of acute pericarditis, do not differ essentially from those which belong to the post-mortem history of other serous inflammations. The appearances vary according to the stage of the disease at which death takes place. Death rarely occurs at the very commencement of the inflammatory action. In some instances in which the disease has proved rapidly fatal, the serous surface has been found more or less reddened, mainly from injection of the vessels situated in the subjacent areolar tissue. The redness is arborescent and in specks or patches, the latter giving to the surface a dotted or mottled aspect. As the sole evidence of inflammation, however, mere redness and vascular injection are not reliable. The latter may be due to various causes which impede the circulation in the heart shortly before, or at the time of, death; and the former may be produced after death by extravasated serum colored with the hæmatin of the blood globules. On the other hand, the redness which belongs to inflammation, here, as in other situations, may have existed during life and disappeared after death. Opacity of the membrane, alteration in its consistence, or the presence of inflammatory products, are essential to constitute proof positive that inflammation has existed. Abnormal dryness of the membrane has been supposed to be an effect of incipient inflammation, and, immediately succeeding this, a glutinous or sticky sensation communicated to the finger when passed over the surface. The latter condition, from its resemblance to that of some fishes when they have been several hours out of water, has been called by French writers, *poissonneux*. These signs, however, as well as vascularity and redness in specks or patches, are not, in themselves, sufficient anatomical evidence of pericarditis. They derive their claim to be included among the anatomical characters of the disease, from their association with the ante-mortem history, and with other post-mortem appearances which are unequivocal in their significance. Acute inflammation speedily leads to the exudation of lymph. This exudation takes place in most cases, probably within a few hours from the commencement of the inflammatory attack. It is, at first, of a jelly-like consistence and adheres slightly to the membrane, forming a thin layer, either limited to the base of the organ and about the roots of the large vessels, or extending, more or less, over the pericardial surface. The heart, at this stage, covered with thin, soft lymph, presents an appearance which has been compared to hoar frost, or to a "layer of liquid gelatine spread upon the parts with a camel's-hair pencil." The exudation goes on, and generally, but not invariably, more or less liquid effusion accumulates within the pericardial sac. If the disease do not prove fatal during this period, the liquid is gradually resorbed, and adhesion of the pericardial surfaces brought into apposition follows.

It suffices to divide the disease into three stages, the division being based on the series of morbid events just mentioned. The brief period during which the membrane is supposed to be dry, or when a glutinous exudation is appreciable by the touch and not by the eye, is by some reckoned as the first or dry stage. Practically, this division is, to say the least, superfluous. The first stage may be considered as extending to the time when the accumulation of liquid is sufficient to be determined during life by symptoms and physical signs. The second stage will embrace the period during which an appreciable amount of liquid continues. The third stage comprises the duration of the disease after resorption of the liquid. These stages have been called, respectively, the stage of exudation, of liquid effusion, and of adhesion; these terms, however, are open to criticism, and a more simple mode is to speak of the disease as consisting of three periods, namely, before, during, and after liquid effusion, the latter expression being understood as applying to a quantity of effused liquid sufficient to distend, more or less, the pericardial sac.

If the disease end fatally during the first period, or before much accumulation of liquid takes place, the heart usually presents a coating of lymph, varying in different cases in its thickness and extent of diffusion. The coating, as a rule, is oftener present and is more abundant at the base than over the other portions of the organ. It may be situated on both the visceral and parietal surfaces of the pericardium, or it may be limited to the former. It is very rarely, if ever, found exclusively on the parietal surface. The lymph is soft, slightly adherent, being very easily removed, and presents, in different cases, diversities of appearance which subsequently become more marked, and will be presently noticed. The membrane is more or less opaque, and may present the arborescent and dotted redness already mentioned. The latter, however, are often wanting after death. In some instances, when the deposit of lymph has been removed, the general aspect of the organ is not, in a marked degree, morbid; in some instances, the membrane covering the heart is studded with prominences resembling the enlarged papillary bodies on the tongue; in other words, it presents a mammillated aspect. The opacity is due to infiltration beneath the membrane; and this infiltration loosens the attachment of the membrane, so that it is detached from the heart with greater facility than in the normal condition of the organ. The exudation within the sac, in some instances, consists almost entirely of lymph, and an amount of liquid sufficient to be determinable does not occur during the progress of the disease. The affection in these cases is analogous to dry pleurisy; and they have been called cases of dry pericarditis. In most cases, however, inflammation diffused over the pericardial surfaces, in other words, general pericarditis does not run its course without giving rise to more or less liquid effusion. When this effect does not occur, the inflammation is generally partial, that is, limited to a circumscribed portion of the membrane.

The accumulation of liquid sufficiently to be manifested by physical signs, takes place at a period from the commencement of the disease varying in different cases. The quantity becomes sometimes large enough to occasion distension of the pericardial sac in twenty-four or thirty-six hours from the date of the attack. In the majority of cases, three or four days elapse before this occurs. As just stated, in some cases it does not take place during the whole career of the disease. The amount of effusion also varies greatly in different cases. From four to six ounces of liquid may be determinable in some cases; and the quantity which may accumulate beyond this amount ranges from a few additional ounces to as many as eight pounds. A case is reported by Corvisart, and also one by the late Dr. Swett,[1] in which the accumulation attained to the maximum just stated. The distension in these cases was enormous, exceeding several times the limit to which the healthy sac is capable of being dilated by forcible injection of a liquid after death. Experiments made by Sibson to determine the latter point showed that, in the adult male at fifty years of age, the injection of twenty-two ounces of liquid dilated the sac to its utmost capacity.[2] Very great accumulation belongs rather to chronic than acute pericarditis. The quantity in the latter very rarely exceeds one or two pints. The effused liquid is more or less turbid.

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[1] Lectures on Diseases of the Chest.

[2] Bellingham, op. cit., Part I, p. 22.

It is sometimes transparent at the surface, resembling clear serum, but muddy and thick at the bottom. The turbidity proceeds from the admixture of lymph, and detached portions of the latter in flocculi or shreds are found in greater or less quantity at the dependent portion of the sac. This turbidity and the presence of flakes of lymph distinguish inflammatory effusion from the transudation which constitutes simple dropsy in this situation, or hydro-pericardium. Occasionally, the liquid effused in pericarditis is sanguinolent, the blood probably being derived from the rupture of vessels in newly-organized structure. Laennec considered this as a variety of pericarditis, which he called hemorrhagic. The admixture of lymph sometimes renders it puruloid; and in some very rare instances the liquid may be truly purulent. In these instances the disease bears to ordinary pericarditis the same relation as empyema to ordinary pleurisy.

In the majority of cases in which acute pericarditis proves fatal *per se*, death occurs during the period of liquid effusion. On examination post mortem, the pericardial sac is found to contain a certain quantity of turbid, puruloid, sanguinolent, or purulent liquid. The free surface of the sac, especially upon the heart, presents, in different cases, diversities of appearance, due to the quantity and disposition of lymph. The deposit is more abundant than it was prior to the liquid effusion. It has become more dense and more firmly adherent to the membrane. It is frequently laminated. Extending, in some cases, over the whole heart, or confined to certain portions, it forms a covering of variable thickness, which often has a reticulated appearance resembling gauze or lace-work; or, quoting comparisons by different observers, the appearance is frequently not unlike that of the section of a sponge, the interior of the gall-bladder, the second stomach of the calf, or a congeries of small earthworms. In other instances the lymph is rolled into ridges, giving to the surface of the heart a furrowed or wrinkled aspect. These ridges are so disposed in some cases as to give rise to an appearance which was compared, poetically, by Hope, to the undulations of sand on the seashore. Another disposition of the lymph is in the form of villous projections, giving to the exterior of the heart a shaggy appearance. In a specimen which I have, the entire surface of the organ is covered with closely set fine filaments from two to four lines in length. This peculiar appearance probably is that to which was applied formerly the name *cor villosum*, or hairy heart. Another variety still is the deposit of lymph in minute patches, thickly disseminated over the heart's surface. The diversities of appearance are explained by the movements of the visceral and parietal surfaces of the membrane upon each other, caused by the systolic and diastolic movements of the heart. They may be rudely imitated, as Laennec remarks, by rubbing together and alternately bringing into contact and separating two marble slabs, their surfaces having been covered with a layer of soft butter. When the pericardial sac is distended with liquid, the two surfaces may come together at certain points during the systole, and recede during the diastole. It is evident that this cannot occur when the pericardial sac is empty; the two surfaces must then be constantly in contact, friction of the surfaces only taking place, without separation. The deposit of lymph, as already stated, may be general or limited to certain situations. It is most prone to accumulate near the base and about the large arteries at their origin. It may be found exclusively or most abundant on either the anterior or posterior surface of the organ. It is rarely very abundant on the parietal surface of the membrane.

When death does not occur during the period of the continuance of an abundant liquid effusion, the latter is absorbed sometimes quite rapidly, and in other cases slowly, and the pericardial surfaces again come into apposition. In examinations after death, at a period more or less remote from this occurrence, these surfaces are found united. Adhesion has taken place. This may be either mechanical or vital, applying the latter term to adhesion by means of newly produced tissue. In some specimens the parietal portion is simply agglutinated to the heart, and, when detached, successive layers of condensed lymph are found to intervene between this portion and the exterior of the organ. These layers may sometimes be peeled off, one after the other, presenting the appearance of distinct, firm membranes. Those nearest the heart are sometimes reddened with hæmatin. Collectively, they form a mass which may be nearly or quite an inch in thickness. This mode of adhesion is purely mechanical. In other specimens newly-organized structure is formed, and adhesion by a vital union takes place. The pericardial sac may be in this way completely obliterated. It was probably cases of this kind which, coming under the observation of some of the old anatomists, led them to conclude that the pericardium was sometimes wanting. Union by an organized attachment may be partial. Sometimes it is limited to certain points, and, the newly organized structure becoming elongated by the movements of the heart, the opposing surfaces are connected by membranous bridles or bands. The pericardial surfaces may become firmly adherent over one side of the heart, and the remainder of the sac contain a considerable quantity of liquid. Examples of this kind have fallen under my observation. In one case adhesion had occurred over the left half of the organ, and the quantity of liquid contained in the right half of the sac was so great as to extend far beyond the right margin of the sternum, giving rise to physical signs which were supposed to denote effusion into the right pleural cavity. In another case, the adhesion was over the right side, and the accumulation of liquid in the left half of the pericardial sac gave rise to a tumor which projected far beyond the left border of the heart. The process of adhesion may be completed within a period varying from a few days to several weeks. The strength of the adhesion is a test of its age. When recent, the attachment is easily broken, but it becomes extremely firm after the lapse of considerable time. A fibroid structure connecting the parietal and visceral surfaces is sometimes one-eighth or more of an inch in thickness.

In examinations after death of bodies dead with various diseases, the symptoms during life not having pointed to the existence of any cardiac affection, one or more opaque patches are often found on the heart, generally situated on the anterior portion of the right ventricle, near the middle or toward the base of the organ. These are the white or milk spots (*maculæ albidæ vel lacteæ*) which have given rise to considerable discussion among pathologists. They vary in size from that of a half dime to a quarter of a dollar. Their form is variable, being round, oval, or irregular in their contour, and sometimes linear. The question is, to what extent are they to be considered as evidence of ancient pericarditis? They consist, in nearly all the instances in which I have examined with reference to this point, of lymph deposited in a thin layer, closely adhering, but which may be stripped off, leaving the surface of the heart beneath normal, except that it has lost somewhat of its naturally smooth and polished appearance. It is stated, however, that in some instances they proceed from opacity and thickening of the membrane itself. That they result from inflammation when they are due to the presence of lymph, must be admitted. They constitute evidence of partial or circumscribed pericarditis, but so limited in extent as not to give rise to any symptoms of disease or any evil consequences. Practically, they are of no importance. They are undoubtedly due to some local cause; and the most probable explanation is that which attributes them to the attrition between the anterior surface of the heart and the thoracic walls. When the membrane itself is thickened, they are probably a kind of callosity caused by pressure. They are rarely found in subjects under the age of puberty, but very frequently after forty; and they are much more common in males than in females.

PATHOLOGICAL RELATIONS AND CAUSATION OF PERICARDITIS.

Acute pericarditis, as an idiopathic or primary disease, is extremely rare. The chances of its development, irrespective of any other disease, in a healthy person, are very few. In this respect it may be classed with acute gastritis and meningitis in the adult. In the vast majority of cases it is a secondary affection. The pathological relations of pericarditis, therefore, form an important as well as interesting portion of the etiological history of the disease. The affections of which it is an occasional concomitant are numerous, but in much the larger proportion of instances it occurs in the course of either acute articular rheumatism or the renal affections known as the different forms of Bright's disease or as Bright's diseases.

The attention of clinical observers has been directed to the occurrence of pericarditis in cases of rheumatism only within the last forty years. The occasional association of these affections was noticed by Pitcairn in 1788, by Dundas in 1809, and by Wells in 1812; but the existence of a pathological relation between them was not fully recognized prior to the publications of Latham and Elliotson in 1829. The subsequent observations of Bouillaud, Hope, and others, established the fact that, in a large proportion of the cases of pericarditis, the disease occurs in connection with rheumatism. Although it is observed much less frequently than endocarditis in this connection, the proportion of the cases of acute rheumatism in which it becomes developed is tolerably large. Of 847 cases collected from various sources and analyzed by Dr. Fuller, it existed in 142, being in a ratio of about 1 to every 6 cases. These cases were reported by six trustworthy observers, and it is worthy of note that each collection of cases gives not far from the same ratio as when they are analyzed collectively.[1] Of 19 cases of recent pericarditis, the histories of which I had recorded when this work was written, in 1859, in 6 the affection occurred manifestly in connection with rheumatism. Of 31 cases subsequently recorded, in 13 the pericarditis was rheumatic. Adding together the two collections, the disease was associated with articular rheumatism in 19 of 50 cases. Statistics show that rheumatism is more likely to become complicated with pericarditis in proportion to the youth of the patients affected. Thus, Fuller deduces from cases reported by different observers that it occurs in a ratio of more than one-third under the age of 15; of a little less than one-fifth between the ages of 15 and 20; of less than one-tenth between 20 and 25; whilst above the age of 25 the ratio diminishes with even greater rapidity. A decided influence, therefore, in the production of the disease, in this connection, pertains to age. It appears also to occur in a larger ratio in females than in males.[2] The liability to pericarditis appears to be greater, other things being equal, in proportion to the acuteness and severity of the rheumatic attack, as denoted by the intensity of the local symptoms and of the febrile movement. It is rarely developed in the subacute form of the latter affection ; the chronic form, and the affection called muscular rheumatism, do not involve a liability of its occurrence, and the same may be said of gout. In one of my cases it was associated with gout, the articular surfaces in different joints being found after death to be incrusted with the urate of soda; but in this case the kidneys were contracted. It has been observed to become developed oftener in the first attack than in subsequent attacks of rheumatism. It may occur at any period during the course of the rheumatic affection, but in the majority of cases it is developed between the fourth and twelfth days. Several instances have been reported in which it preceded the affection of the joints; I have met with an instance of this kind. In a case which came under my observation it occurred coincidently with an affection of the wrist, and in another case at the same time that a knee and a shoulder joint were affected. In a case observed by Fuller the pericarditis occurred five days before any of the joints were affected. According to West, it oftener takes precedence of the affection of the joints, and is more apt to occur coincidently with, or shortly after, the latter, in children than in adults.[1]

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[1] On Rheumatism, Rheumatic Gout, and Sciatica. By Henry William Fuller, M.D., &c. Am. edition, 1854, p. 216.

[2] Fuller, op. cit

What is the nature of the pathological relation existing between pericarditis and acute rheumatism? It was formerly supposed that a transference of the disease from the joints to the heart took place, and that the pericarditis was due to change of seat, or metastasis. This supposition is disproved by the fact that the inflammation of the pericardium does not involve necessarily, nor indeed, generally, diminution of the articular inflammation. The reverse of this obtains in the majority of cases. Moreover, as just stated, the pericarditis in some instances precedes the affection of the joints. The same internal morbid condition which determines the latter, gives rise to the former. The one, as well as the other, is the local expression of a general or constitutional affection, involving, probably, blood-changes in which consists the essential pathology of rheumatism. The pericarditis and the affection of the joints, in other words, are, alike, effects of a common pathological condition.

To inquire respecting the nature of the blood-changes which constitute this condition, does not fall within the scope of this work. As the affection of the joints in rheumatism exists in the larger proportion of cases without the development of pericarditis, so it is probable that rheumatic pericarditis may sometimes occur without the former. The liability of the pericardium to become the seat of inflammation in rheumatism, is to be explained by the analogy of structure between this membrane and the tissues entering into the composition of the articulations.

Since our knowledge of the connection of albuminuria and uræmic phenomena with certain affections of the kidney dates from the researches of Bright, published in 1827, it follows that the pathological relation existing between pericarditis and these affections had not previously been ascertained. Clinical observation within late years has abundantly established the existence of such a relation. Of 35 cases of pericarditis analyzed, with respect to causation, by Dr. John Taylor, renal disease existed in 13, and the development of the pericardial inflammation could not otherwise be accounted for.[1] Of 19 cases taken from my records, renal disease was present without any other apparent causative agency, in 3, and probably, also, in 2 additional cases. This was true of 2 out of 31 cases analyzed in preparing the second edition of this work. Disease of the kidney existed in more than two of these cases, but in the others there were complications on which the pericarditis may have been dependent. On the other hand, of 50 patients who had either died of renal disease, or who were ascertained to have disease of the kidneys in an advanced form, acute pericarditis was found by Taylor in 5, or in the ratio of 1 to 10. Of 292 cases of renal disease analyzed by Frerichs, pericarditis occurred in 13.[2] Of 135 fatal cases of pericarditis analyzed by Dr. T. K. Chambers, the kidneys were diseased in 36.[3] Renal disease exists in a larger proportion of the cases of pericarditis which end fatally, than of those ending in recovery. The explanation of this is, pericarditis developed in connection with Bright's disease, especially at an advanced stage of this disease, almost invariably ends fatally; whilst in connection with acute rheumatism, recovery takes place in a large proportion of instances.

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[1] On Diseases of Children, second American edition, p. 304.

What is the nature of the pathological relation existing between pericarditis and the renal affections generally included under the name Bright's disease or Bright's diseases? Clinical observation shows that, in connection with the latter, serous inflammations are apt to become developed. The production of these inflammations, as well as other effects, are attributed to the accumulation of urinary principles in the blood, in consequence of the impaired excretory function of the kidneys. The intermediate morbid condition determining the pericarditis, is thus supposed to be uræmia. The urea in excess, or the products of its decomposition in the blood, act as poisonous agents, giving rise to inflammation of the pericardial and other serous membranes, together with other pathological consequences. This is the explanation which is most consistent with our present knowledge.

Pericarditis is not very infrequently associated with either pleurisy or pneumonia. It has been inferred from this association that the inflammation extends from the pulmonary organs to the heart, in consequence of the proximity of the latter to the former, an inference which appears to be strengthened by the fact that the coexisting inflammation of the pulmonary structures is situated on the left, oftener than on the right side. But pleurisy and pneumonia are diseases of frequent occurrence, and in the great majority of cases the inflammation does not extend to the pericardium. On the other hand, in cases of pericarditis developed in connection with acute rheumatism, the inflammation very rarely extends from the heart to the adjoining pulmonary structures. It is chiefly in non-rheumatic cases of pericarditis that this disease is associated with pulmonary inflammation. It is, therefore more rational to conclude that between the latter and the former there exists no relation of causation, but that both are equally dependent on some internal, determining pathological condition. This condition belongs, in a certain proportion of cases, to disease of the kidneys, and, in other cases, to some of the diseases occasionally giving rise to pericarditis, which are presently to be noticed. Although it is true that when pleurisy is associated with pericarditis the left pleura is oftener affected than the right, yet the right pleura is not infrequently the seat of the inflammation. Of seven fatal cases, of which I have preserved notes, the right side was affected in three. The pleuritis was double in three of these cases.[1] To show the relative frequency of the occurrence of pleurisy and pneumonia in cases of non-rheumatic as compared with rheumatic pericarditis, the following statistics by Dr. Taylor may be cited: In 24 cases of non-rheumatic pericarditis, pneumonia was associated in 12, while it coexisted in only 4 of 16 cases of rheumatic pericarditis. Pleurisy was associated in 10 of 16 cases of the former, and in only 7 of 24 cases of the latter. Of 31 cases analyzed in preparing a second edition of this work, pneumonia was associated in 9 cases, and chronic pleurisy in 1 case. The pneumonia was double in 1 case; the right lung was affected in 4, and the left lung in the same number of cases. In only one of these cases was the patient afflicted with rheumatism.

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[1] On Some of the Causes of Pericarditis. Medico-Chirurgical Transactions, vol. xxviii, p. 453.

[2] N. A. Med.-Chir. Rev., Nov., 1860.

[3] Decennium Pathologicum.

Pyæmia appears to rank next to Bright's disease as regards the frequency of its coexistence with pericarditis in fatal cases of the latter. It existed in 18 of the 135 subjects examined by Chambers, a ratio equal to that of rheumatism in this collection of cases. Pyæmia, at the same time, is likely to give rise to inflammation affecting serous structures in other situations. It is probably through the intervention of this blood affection that wounds of parts remote from the heart and surgical operations sometimes give rise to pericarditis. Of the nature of the pathological relation existing between pyæmia and pericarditis all that can be said with our present knowledge is, that the blood is so altered as to determine, among other results, inflammation of the pericardium.

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[1] Louis' statistics show coexisting pleuritic or pneumonic inflammation to be limited to the right side in one-third of the cases in which these pulmonary affections are associated with pericarditis.

The eruptive and continued fevers occasionally become complicated with pericarditis. It is extremely rare for this complication to occur in connection with either typhoid or typhus fever; but its development in the course of scarlatina and small-pox is not very uncommon. When it occurs as a sequel of scarlatina, of which I have met with an instance, it is probably dependent on the morbid condition incident to albuminuria. In the instance just alluded to, it was preceded by general dropsy and albuminous urine. The pathological relation with the essential fevers, also, involves certain internal causes pertaining probably to blood-changes.

Pericarditis has been observed to occur in cases of scorbutus. Its occurrence in this pathological connection was observed some years since during the prevalence of scurvy among sailors at St. Petersburg by M. Seidlitz, of that city; and a variety of the disease, said to be frequent on the extreme northern coasts of Europe, where scurvy is endemic, has been described by Kyber under the name *pericarditis scorbutica.* Another observer, Karawagan, found that of sixty subjects dead with scurvy, thirty were affected with pericarditis. As described by these three writers, the pericarditis occurring in scurvy differs from the ordinary form of the disease, in the bloody character of the liquid effusion contained in the pericardial sac. It is, in fact, a species of hemorrhagic pericarditis.[1]

Purpura and cyanosis are other affections, characterized by a morbid condition of the blood, in connection with which pericarditis has been observed to become developed. It is, however, doubtful if the number of instances in which the association occurs is sufficient to show the existence of any special pathological relation.

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[1] Bellingham, op. cit., pt. ii, p. 263.

Tuberculosis of the lungs is sometimes associated with pericarditis. Of eleven fatal cases in which the pericarditis was recent, pulmonary tuberculosis coexisted in three. This association is probably due merely to coincidence. The tuberculous cachexia does not appear to give rise to inflammation of the pericardium, except in the very rare instances in which tubercle is deposited upon this membrane. The same remark is applicable to the carcinomatous cachexia.

As thus far considered, the causation of pericarditis has, for the most part, involved internal morbid conditions pertaining to certain general diseases, cachexiæ, and blood-changes. In the great majority of cases, the disease is developed secondarily in some of these pathological relations, and, more especially in connection with acute articular rheumatism. But various local causes may give rise to the disease. It may be produced by wounds penetrating the pericardial sac, and by other injuries of the chest. In one of my cases the pericardium had been wounded by the tine of a fork. The patient fainted shortly afterward, and kept the bed, suffering much from pain for three days, when he walked ten miles, and took passage from Vicksburg to New Orleans. On entering the Charity Hospital at New Orleans he was found to have pericarditis, from which he rapidly recovered. A very remarkable case of traumatic pericarditis occurred at Bellevue Hospital. A patient was admitted into the hospital in a state of alcoholic insensibility. On emerging from this state he declared that he had swallowed a plate with false teeth attached to it. This statement was not credited, but death occurring some time afterward, the autopsy confirmed its correctness. The plate had lodged in the œsophagus, and had made its way by ulceration in part within the pericardial sac, giving rise to pericarditis. The specimen was preserved and is in the museum of the hospital. Abscesses formed in the liver occasionally open into the pericardial sac and occasion acute inflammation. Collections of softened tubercle in the lungs have been known to take this direction. Mediastinal abscesses may pursue the same course. The pericardium sometimes appears to take on inflammation in consequence of the local irritation excited by aneurismal tumors of the aorta. The deposit of either tubercle or carcinomatous matter upon this membrane, happily extremely rare, is another local cause to which allusion has already been made.

Pericarditis may occur as a primary or idiopathic affection. Cases, however, are so extremely rare that clinical observers of large experience declare they have never met with an example. On this point, Walshe remarks that "alleged idiopathic pericarditis becomes rarer every year, in proportion as the evolution of diathetic diseases grows more fully understood;" and he adds that he has never seen a positive case of the kind. A case which I observed in the New Orleans Charity Hospital, in December, 1860, affords an illustration. The patient, a male, aged 21, had been in hospital with intermittent fever. After having been discharged, he was about the streets looking for work, and was seized with pain in the centre of the sternum. He returned to the hospital, and presented the signs of pericarditis with considerable effusion. There were no manifestations of articular rheumatism, the urine was free from albumen, the chest organs, other than the heart, gave no morbid signs, and, in short, pericarditis alone existed. He was discharged recovered after having been fifteen days in hospital.

The influence of youth in the causation of rheumatic pericarditis has been referred to. Statistics show that non-rheumatic cases embrace a larger proportion of persons beyond the middle period of life. The average age in twenty-four cases of the latter, reported by Ormerod, was forty-two; while in sixty-one cases of the former, the average age was twenty-one. But no period of life is positively exempt from the liability to pericarditis. Numerous cases have been reported in which the disease occurred in infants but a few months old. In some of these cases the most prominent symptom was screaming of the infant. I have met with a case of the disease in a child eight months old, in which this was the chief symptom, the sudden, sharp, brief cry resembling that which is characteristic of meningitis, and leading to a suspicion of the existence of the latter affection.

In a large proportion of cases of pericarditis, endocarditis coexists. In rheumatic pericarditis this is a rule to which there are few, if any, exceptions. The rule does not hold good, at least to the same extent, in non-rheumatic pericarditis. The frequent association of the two affections will serve, in a measure, to account for the fact that when, on examination after death, the evidences of ancient pericarditis are discovered, the heart is often, if not generally, more or less enlarged, and valvular lesions are, at the same time, found. The remote effects of pericarditis on the heart will be considered in connection with pericardial adhesions.

SYMPTOMS OF ACUTE PERICARDITIS.

The symptoms of acute pericarditis vary according to the intensity of the inflammation, the amount of liquid effusion, and other circumstances, the effects of which are sufficiently manifest. But, irrespective of these, variations in different cases are observed, which cannot be traced to obvious differences in the morbid conditions pertaining to the organ affected. In this respect, however, the disease does not differ from other inflammations, especially those affecting serous structures. The same is true of pleuritis, meningitis, and peritonitis. Certain of the symptomatic phenomena, such as pain, febrile movement, &c., are present in a marked degree in some cases, in a moderate or slight degree in other cases, and they are sometimes wanting when the appearances after death denote an equal intensity and extent of the inflammation. There is, in short, often an apparent want of correspondence between the manifestations of the disease during life, and the changes ascertained after death, showing that the symptomatic events which belong to the clinical history of the disease are influenced, in no small measure, by circumstances pertaining to other parts of the body than the organ affected, or to the general system. These circumstances are but little understood; but this remark is not more applicable to pericarditis than to various other local affections. This frequent want of harmony (if this term may be allowed) between local morbid conditions and symptomatic phenomena, is important to be considered in connection with diagnosis, prognosis, and treatment. It is this which invests the physical signs of disease, whenever they are available, with much of their great practical value. Pericarditis being associated, in the great majority of cases, with other affections, its own manifestations are, to a greater or less extent, intermingled with, and obscured by, those of the latter. This fact, together with the variations just alluded to, impairs considerably the general application of a descriptive history based on the clinical study of the comparatively rare cases in which the disease is isolated and its symptomatic phenomena strongly marked.

The division of the career of the disease into three periods, based on facts pertaining to its morbid anatomy, is to be borne in mind. The symptoms undergo important modifications when an abundant accumulation of liquid takes place in the pericardial sac, in other words, during the second period; and, again, after absorption of the liquid, or during the third period.

In treating of the symptomatology of pericarditis, the symptoms which relate directly to the heart will be first noticed, and, afterward, those referable to different anatomical systems —the circulatory, respiratory, nervous, &c.

Symptoms referable directly to the heart.

The symptoms which relate directly to the heart, are pain, palpitation, and tenderness.

Pain referred to the præcordia is a prominent symptom in some cases. The character of the pain is burning or lancinating, and it is often accompanied by a sense of constriction. It is aggravated by inspiration; and the inspiratory acts are sometimes shortened in consequence, the number of respirations per minute being correspondingly multiplied. The pain is also increased by movements of the body. It may be referred to the region of the heart or to the epigastrium. It is sometimes referred, at first, to the right side, or to the centre of, the sternum. It may extend to the back, to the left shoulder, and down the left upper extremity, bearing some resemblance, if severe, to the pain in angina pectoris. The character of the pain being the same in some cases as in pleurisy, the affection is liable to be mistaken for the latter. Pleurisy, as has been seen, is, in some cases, associated with pericarditis, and the pain belonging to each affection separately, cannot, under these circumstances, be readily disconnected. The pain, also, is not unlike that incident to pleurodynia and intercostal neuralgia, and these affections may coexist with pericarditis. But pain is by no means invariably a prominent symptom with pericarditis. In the majority of cases it is either moderate or slight in degree. It may be wanting. In a case in which pericarditis was consecutive to pleurisy of the left side, with large effusion, the heart being removed to the right of the sternum, no pain was experienced in the organ at the time of the attack, or subsequently. In another case the pain was slight, and referred to the region of the dorsal vertebræ. In the larger number of the cases which I have observed the pain has been either slight or moderate. I have rarely witnessed excruciating suffering from pain in connection with pericarditis. Pain, thus, is variable in degree; it is not reliable as a constant symptom, and, when more or less marked, it is not distinctive.

Upon what does the occurrence of pain depend? Bouillaud attributes it, in all cases, to coexisting pleuritis. But it may undoubtedly be present, and prominent as a symptom, when pleurisy does not coexist. It must emanate from the nerves of the heart; and, although it is difficult to explain the differences in the amount of pain in different cases which appear to be similar as regards the intensity of the inflammation, and to account for the absence of pain in certain cases, it is to be considered that the same difficulty is met with other serous inflammations, for example, peritonitis. The increase of pain during the act of inspiration is explained by the friction of the pericardial surfaces in consequence of the depression of the diaphragm, and by the pressure, upon the heart, of the lungs in the process of inflation. When pain is present, it belongs especially to the commencement of the attack and the early part of the disease. It diminishes or disappears when the inflamed surfaces become covered with lymph, and are separated by liquid effusion. At this stage it may give place to a sense of uneasinesss or undefined distress referable to the præcordia, not amounting to positive pain.

Tenderness on pressure is a symptom generally, but not constantly present. Like pain, when present, it is variable in degree. It is rarely very marked. As pointed out by Hope, tenderness may sometimes be discovered on pressing upon the epigastrium beneath the cartilages of the ribs in a direction toward the heart, when it is not apparent in the præcordia directly over the heart. In order to constitute a symptom of pericarditis, it must be limited to the region of the heart. It is needless to say that when pleurisy, affecting the left side, coexists, the tenderness will be diffused, more or less, over the whole of that side. In acute rheumatism, pleurodynia may occur, either with or without pericarditis, and diffused tenderness will then be present. In rheumatic pericarditis, I have found the whole of the left side exquisitely sensitive to pressure, without the physical evidence of pleuritis. As an isolated symptom, circumscribed tenderness is of little value, but, taken in connection with other symptoms, it is of importance. It is to be borne in mind that the absence of tenderness is not positive evidence against the existence of pericarditis.

Tenderness is doubtless due to an abnormal sensibility developed in the inflamed membrane. Why this sensibility is present in some cases, and not in others, cannot be explained; but the same is true of other serous inflammations. In peritonitis, for example, the tenderness is generally great; but in a well-marked case at this moment under observation, this symptom is wanting.

Increased action of the heart is an effect of pericardial inflammation during the early part of the disease. The contractions are violent and sometimes irregular. The patient is conscious of an unnatural beating of the organ. This constitutes palpitation. Like the other symptoms referable to the heart, this is by no means constant, and it possesses, in itself, but little value, inasmuch as it occurs in connection with the different forms of organic disease, and, also, as a purely functional disorder. Its importance depends on its association with other symptoms. It is sometimes strongly marked. The commencement of the disease may be characterized by tumultuous action of the heart. In cases of acute rheumatism, this should excite strong suspicion of pericarditis, and lead to a careful examination for more positive evidence of the disease. Palpitation belongs to the first stage of pericarditis. It is incompatible with much liquid effusion, and even if the latter do not occur, a secondary effect of the inflammation on the muscular substance of the heart, is diminished power of contraction, or incomplete paralysis. A similar effect is observed in cases of peritonitis, as shown by enlargement of the intestines from the pressure of their gaseous contents.

Symptoms referable to the circulation.

The pulse, considered alone, in this, as in most diseases, does not furnish characters which, in a diagnostic point of view, are highly distinctive; but, the diagnosis being made, it gives important information respecting the condition of the heart. At the onset of the disease, it corresponds to the increased muscular action of the organ, and is frequently strong, quick, vibratory, as well as more or less frequent and sometimes irregular. In proportion as the heart is weakened, in the progress of the disease, it becomes enfeebled; and when, in conjunction with a certain amount of paralysis, the movements of the organ are mechanically restrained by the pressure of liquid effusion, the pulse is notably small and weak, with more marked disturbance of its rhythm. It represents, thus, the effects of the disease, vital and mechanical, on the circulation. Stokes thinks that the effect upon the heart's movements produced by the pressure of liquid in the pericardial sac is overrated, and he cites, in support of this opinion, the comparatively small disturbance of the circulation caused by dislocation of the heart in cases of empyema. But the condition of the organ in the two cases is by no means the same. When the heart is removed to the right of the sternum by the pressure of a large accumulation of liquid in the left pleural sac, the freedom of its movements is but little restrained in comparison with the effect of an abundant effusion within the pericardial sac. When the latter is distended to double or treble its normal capacity, it is truly surprising that the cavities receive sufficient blood for the circulation to be carried on. The fact that the circulation is not arrested shows the force with which the blood is returned to the heart. In dwelling upon the atony or paralysis arising from the proximity of the inflamed membrane to the muscular tissue, Stokes seems to me to undervalue the mechanical effect of the presence of liquid. The latter effect is proportionate, other things being equal, not so much to the amount of liquid effusion as to the rapidity with which it takes place. If it accumulate slowly, the dilatation of the sac goes on *pari passu*, and the heart, speaking metaphorically, becomes accustomed to the pressure. If the quantity, on the other hand, become rapidly large, the sac does not readily yield, and the heart suffers from the compression in a more marked degree. Clinical observation sustains the correctness of these remarks. The pulse may be greatly enfeebled from the weakness of the heart induced as a secondary result of the inflammation, irrespective of effusion. But the pressure of liquid, especially when rapidly effused, affects the pulse to a still greater extent. A weak and small pulse belongs to the stage of effusion, and may be considered as representing, in a great measure, the extent to which the heart is mechanically restrained. As regards frequency of the pulse, Walshe remarks that it "is subject to more sudden variations from the influence of emotional excitement and effort than in any other disease, perhaps." He adds that he has known a very gentle movement of the trunk raise the pulse from 80 or 90 to 130 or 140. It is often found to vary notably on different days, without any obvious cause.

During the progress of pericarditis, then, as a rule, the pulse is at first more or less increased in frequency, and also in force and quickness; and, afterward, from the combined effects of diminished muscular power and the pressure of liquid effusion, it is irregular, weak, and small, the frequency being generally still more increased. But to this rule there are exceptions. The frequency during the first stage is, in some cases, not greater than in health. Graves, indeed, states that he has observed it to be less frequent than in health. It may continue regular during this stage, and present no marked deviation from its normal characters. During the second stage, the effect of even a large accumulation of liquid is sometimes not very marked. It may retain considerable force and volume when the extinction of præcordial impulse and other physical signs show distension of the pericardial sac.

In cases of rheumatic pericarditis, the pulse is more or less accelerated prior to the development of the heart affection. The influence of the latter, therefore, cannot be estimated with precision. A sudden change in frequency, or other characters, during the course of acute rheumatism, occurring when no joints are newly attacked, and irrespective of any obvious cause, should lead the practitioner always to direct his attention to the signs of cardiac disease.

The obstruction to the circulation incident to prolonged accumulation of liquid in the sac may be sufficient to give rise to œdema of the lower extremities and face. In general, however, œdema involves coexisting disease of the kidneys, or organic lesions of the heart.

Lividity of the lips, face, &c., may be due alone to the pressure of liquid and weakness of the heart. As a symptom of pericarditis, exclusive of other affections, it belongs to the second stage of the disease, and denotes an alarming degree of obstruction. The pulse will be found to be, at the same time, extremely feeble and irregular. The lividity, under these circumstances, depends on congestion of the venous radicles, arising from inability of the heart to receive the blood returned to it by the systemic veins. But this symptom generally involves an affection of the pulmonary system, such as pleuritis or pneumonitis, existing in combination with the pericarditis. The impaired ability of the lungs to aerate the blood is then associated with venous congestion in the production of lividity. It is unnecessary to add that lividity is a symptom incident to various cardiac and pulmonary affections, and does not therefore possess intrinsic significance as diagnostic of pericarditis. It is not, in fact, always present in fatal cases of the latter disease, even when they are characterized by great liquid effusion. These remarks are equally applicable to œdema.

Symptoms referable to the respiratory system.

In cases of pericarditis disconnected from any affection of the lungs, the respirations are sometimes accelerated in consequence of the inspiratory acts being shortened by præcordial pain. Marked dilatation of the *alæ nasi* may be observed under these circumstances. Cough, dry, hacking, or spasmodic, is common, and does not denote, necessarily, coexisting pulmonary disease. Dyspnœa may be an urgent symptom, dependent generally on congestion of the lungs incident to compression of the heart by liquid effusion. These symptoms, however, are extremely variable and inconstant, as are all the symptomatic events belonging to the clinical history of pericarditis. The respiration may be unaffected, or accelerated only in proportion to the febrile movement. Cough is not uniformly present. Dyspnœa may be wanting even when the pericardial sac is largely distended. Moreover, each of these symptoms occurs in a variety of pathological relations, and is not, therefore, distinctive of pericarditis. They may be dependent, to a greater or less extent, on coexisting pleurisy or pneumonia. When these affections are excluded, dyspnœa denotes, as a rule, obstruction to the circulation caused by weakness of the heart or mechanical compression, or both combined. As thus produced, it may exist in a degree to constitute orthopnœa. It belongs, in general, to the second stage of the disease, and is associated with frequency and feebleness of the pulse, and perhaps with lividity of the prolabia and face. Under these circumstances, it denotes imminent danger. Cases, however, have been observed in which dyspnœa, or even orthopnœa, existed at the commencement of the disease, prior to effusion, and when it was not attributable to any coexisting pulmonary affection. In some instances, as suggested by Sibson, the distended pericardial sac may add to the dyspnœa by pressing on the trachea at its bifurcation. The augmented space which the pericardial sac occupies when largely distended also contributes to the production of dyspnœa.

The voice has been observed to be remarkably weak, the patient being unable to speak in feeble tones without considerable effort. Walshe, who has noticed this symptom, states that it seems to be mainly connected with copiousness of effusion.

Symptoms referable to the digestive system.

Certain cases of pericarditis are characterized by prominent symptoms referable to the digestive system; but, occurring only occasionally, they are incidentally connected with the disease, and can hardly be considered as forming a part of its clinical history. Thus, vomiting is sometimes present and persisting in a marked degree. Copland has remarked that, under these circumstances, there is some liability to mistake the disease for gastritis, the rapid, weak, irregular pulse, &c., being attributed to a tendency of the latter affection to an unfavorable termination. Dysphagia is another symptom belonging in this category. Its occasional occurrence in cases of pericarditis was noticed by an Italian author, Testa, who published a work on diseases of the heart in 1811.[1] Stokes and Walshe have observed it in several cases. It is noted in one of the cases which I have recorded. It has not been ascertained to depend on any appreciable alteration in the pharynx or the adjoining parts, and is therefore to be regarded as either a spasmodic affection, or a mechanical effect of pressure of the distended pericardial sac upon the œsophagus.

These are the only symptoms to be mentioned under this head.

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[1] *Vide* Stokes on Diseases of the Heart and Aorta, Am. ed., p. 69.

Loss of appetite, thirst, constipation, &c., are incident to pericarditis as well as to acute inflammation affecting any important organ, and accompanied by febrile movement.

Symptoms referable to the countenance, position, &c.

An expression of anxiety or apprehension is frequently a marked symptom. In severe cases, near the fatal termination, the *risus sardonicus* has been observed. Lividity and œdema are occasional symptoms which have been already mentioned.

The position assumed by the patient is generally on the back; or diagonally between that on the back and on the side. The decubitus is rarely on the left side, the liver in this position pressing upon the heart, and giving rise to discomfort or adding to the distress. In some cases a position on the right side is not uncomfortable. If pleurisy or pneumonia coexist, the position will, of course, be in a measure determined by these affections. Generally, but not invariably, the patient desires to have the head and shoulders raised.

All observers have noticed this point relating to position, viz., whatever may be that selected by the patient, he is reluctant to change it; that is, he desires, as much as possible, to maintain the same position. This is accounted for by the fact that movements of the body increase distress, and, by exciting the heart, give rise to a sense of syncope, especially when the pericardial sac is distended with liquid. Fatal syncope may be induced by a change of position. I have known death to occur suddenly, when a fatal termination was not expected, apparently being caused by the patient rising from bed and going to stool.

When the amount of liquid effusion is large, a recumbent position on the back may still be preferred; but sometimes patients experience relief from lying on the face. If dyspnœa be urgent, a sitting posture may be alone tolerable, the dyspnœa then constituting orthopnœa. Under these circumstances, restless movements of the arms are common, the body remaining comparatively immovable.

Symptoms referable to the nervous system.

Mental aberration, moderate or slight, and transient, is not uncommon in cases of pericarditis. I have known it to occur at the commencement of the disease, and soon disappear, the patient afterward preserving the faculties of the mind to the close of life. It is oftener observed at a later period in fatal or severe cases. It is not, however, an element of the disease. In the majority of cases it is wanting. But in certain cases cerebral symptoms are developed which are highly important in themselves, and also because they serve to mask the local symptoms of cardiac disease. The cerebral symptoms now referred to resemble those which characterize different affections of the nervous system. Inflammation of the meninges of the brain, mania, dementia, coma, epilepsy, tetanus, and chorea may be simulated in cases of pericarditis, the latter disease being generally overlooked before death, and examination post mortem revealing no appreciable lesions of the brain or spinal cord adequate to explain the phenomena observed during life. The phenomena in these remarkable, and, as they have been justly called, fearful, cases must needs be diversified in order to give rise to a resemblance to each of the several affections just named; yet there are certain features which are somewhat distinctive. This subject has been considered more fully by Burrows than by any other author within my knowledge.[1] Burrows gives a synopsis of all the cases that he was able to gather from various sources. It is remarkable that the subject has been, in general, only alluded to in most works devoted to diseases of the heart. Without detailing the cases which have been reported, I shall refer to them sufficiently to present a sketch of the varied symptoms which they embrace; and I shall add a brief account of three striking cases which have fallen under my own observation.

The first recorded case, according to Burrows, was reported by Stanley in 1817. The next case was communicated by Abercrombie in 1821. Latham reported a case in 1828; and he states that "when he first related the particulars of his case to several medical friends, they looked incredulous, or rather contemptuous of the man who would mistake an inflammation of the pericardium for an inflammation of the brain." In each of these cases the patient was supposed to labor under a cerebral affection, to which the treatment was directed, and the existence of cardiac disease was not suspected prior to the autopsical examination.

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[1] On Disorders of the Cerebral Circulation, and on the Connection between Affections of the Brain and Diseases of the Heart By George Burrows, M.D., &c , Am. edition, 1848.

The brain presented no evidence of disease beyond a certain amount of congestion. Other cases were subsequently reported by Andral, Bouillaud, Copland, McIntosh, McLeod, Hawkins, Bright, Watson, and others. Burrows states that not less than six cases came under his own observation. Of 16 recorded cases cited by the author last named, 11 proved fatal, and only 5 recovered. Of the 11 fatal cases, in 2 only was an affection of the heart detected during life, in 1 cardiac disease was suspected, but in the remaining 8 cases there was no suspicion of an acute affection of the heart until it was revealed by an examination after death. Of the 5 cases ending in recovery, in 4 the diagnosis of cardiac disease was satisfactorily established.

In the sixteen cases detailed in Burrows's work, were manifested, delirium, convulsions, agitation of the limbs resembling chorea, a state of dementia, a species of coma, seizures resembling apoplexy and tetanic spasms. The delirium was characterized by taciturnity and maniacal excitement under the influence of delusions involving the idea of having committed some crime. Convulsions occurred in paroxysms, and the choreic form was accompanied by rolling of the eyes and head, as well as violent agitation of the limbs. The coma was characterized by the eyelids remaining open, and the eyes fixed. In the apoplectiform seizures, the eyeballs were turned upward and the limbs paralyzed. In several instances, violent tonic spasms occurred, resembling tetanus. In the fatal cases, death was generally preceded by ordinary coma.

The first of the cases which have fallen under my observation, occurred in 1849, and was reported by me, February, 1850.[1] The patient was admitted into hospital in a state of active delirium, and nothing was ascertained respecting the previous history. On the following day he was tranquil, and, when spoken to, made no reply, shaking his head. The eyes had a wild, staring expression. He could not be made to protrude the tongue. The pulse was small, feeble, and not accelerated. Active delirium occurred at intervals, during which he shouted and cried, as if from apprehension of danger. At other times he lay with his eyes open and fixed in a particular direction, taking no notice of persons and things around him. On several occasions he answered questions, and he then gave evidence of the delusion that he had committed some crime. Once, when asked how he was, he replied, "Guilty." At another time he had an impression that he was confined in jail, and subsequently he asked why he had not been hung, &c. On the fourteenth day after his admission, he complained, for the first time, of pain in the chest, and, on physical examination, the signs of pneumonia were discovered. There were no cough and expectoration. Death occurred on the seventeenth day. Delirium with hilarity occurred on the last day, and he became comatose for several hours before death. On examination post mortem, the left lung was in the second stage of inflammation, and the pleural sac contained about twelve ounces of turbid serum. The surface of the heart was covered with recent lymph. The endocardial membrane was healthy and the valves were sound. The brain presented no other evidence of disease than a considerable amount of congestion, and slight opacity of the arachnoid over the superior surface of the cerebrum.

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[1] Buffalo Medical Journal, vol. v, p. 505.

The existence of pericarditis was not suspected, in this case, prior to the autopsy. Up to the fourteenth day, the affection was supposed to be exclusively cerebral, no symptoms pointing to the chest as the seat of the disease. Physical exploration was neglected until the date just stated, when pneumonia was ascertained.

The second case came under observation in the hospital at Buffalo, in 1851, and was reported by me in January, 1854.[1] At my morning visit I found that on the previous evening a patient had been admitted greatly prostrated and delirious. Nothing was obtained relative to the previous history. The patient had not spoken since his admission. He lay with his eyes open, fixed, most of the time, in one direction, taking no notice, and making no reply to questions. A disagreeable peculiarity in this case was, the patient frequently ejected saliva with force, and without any regard to its destination. His bed and the floor were bespattered with spittle. Persons in proximity to him were liable to receive it on their persons, not from design, but because it was scattered at random, the patient not changing his position and lying on his back. Under these circumstances, an examination of the case was deferred, and at my next visit I found that the patient had died. At the time I was observing the patient, the idea of pericarditis did not occur to me, but in thinking of the case afterward, a resemblance in the character of the delirium to that of the preceding case, led me to suspect this disease; so that, before the autopsy was made, I ventured to predict that it would be discovered. My prediction proved true. The pericardium was universally adherent by recent tender adhesions. Pulmonary disease did not coexist in this case.

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[1] Buffalo Medical Journal, vol. ix, p. 449.

The foregoing cases ended fatally. The third case came under observation in the Louisville Marine Hospital, October, 1853, and terminated in recovery. This case was reported in connection with the preceding case. When admitted the mind of the patient was too dull to give any connected account of past or present symptoms. On the day following he was delirious, frequently getting out of bed, and seemed bewildered. The next day he was unconscious. He lay on his back taking no notice of persons and things around him. He had lost one eye; the other remained open, and the pupil was dilated. He was taciturn, and could not be made to reply to questions; he urinated in bed; the saliva escaped from the mouth, and he did not swallow when drink was introduced. The physical signs, exclusive of friction-murmur, were sufficient to establish the existence of pericarditis with moderate effusion.

Convalescence was established a fortnight after his admission. For three days he took neither drink nor nourishment, making no effort to swallow, and sometimes resisting their introduction by forcibly closing the teeth. At times during the three first days he was exceedingly restless, throwing himself from the bed, so that it became necessary to transfer him to the floor. The pulse was 80, and the respirations 28. On the fourth day, in the morning, there was marked improvement. The patient took food and drink, and appeared to notice objects around him. This peculiarity was observed, viz., he directed his vision to some point, now a portion of the pillow and now his hand, protruded his tongue towards it, and then slowly grasped it with his lips and teeth. This he repeated frequently. In the course of the day he again became restless, throwing himself about, getting up, calling names of different persons. The day following, his expression was idiotic. His eye was open, and he looked about with a vacant stare. He resisted physical exploration. Twice he said while the record of symptoms was being made, "I beg pardon." These words were uttered spontaneously, with slowness and hesitancy. He did not reply to questions, and was taciturn the greater part of the time. On the sixth day he had three attacks of convulsions an hour in duration. These recurred on the day following. In the intervals he frequently got out of bed and endeavored to break the walls of the room, as if to escape from persons threatening violence. It was necessary to apply a restraining jacket. On the eighth day he lay night and day rolling about the floor and shouting incoherent words. On the tenth day he continued wakeful, shouting, with occasional manifestations of hilarity. In the course of this day he slept quietly for several hours, took food and drink readily, protruded the tongue, and replied to questions. From this time he was rational, and, on being questioned, said that he had pain in the left breast above the nipple, lancinating, and increased by deep inspiration. He had no recollection of the events of the previous fortnight. He stated that he was ill for two days before coming to the hospital, and that he suffered chiefly from pain in the left breast. He convalesced from this date.

The physical signs on which the diagnosis was based in this case were flatness on percussion over an increased and a pyramidal space in the præcordia; elevation of the point of apex impulse and flatness below this point; irregularity and feebleness of the heart's contractions; diminution of the area of præcordial dulness at the time of convalescence, and afterward retraction of the intercostal spaces with the heart's action. Friction-murmur was not discovered, but the early application of a blister over the præcordia interfered somewhat with auscultatory exploration. In connection with the physical signs denoting pericarditis, pain referred to the præcordia was a prominent symptom before the patient entered the hospital, and was felt after his consciousness returned, together with tenderness in the same region, evidently not dependent on the blister. Acute pulmonary disease was excluded by the absence of physical signs pertaining to the lungs.

It is worthy of remark that in the three cases of pericarditis associated with cerebral disorder, of which an account has just been given, the disease in each was not developed during the course of rheumatism. Of the sixteen cases analyzed by Burrows, in seven no rheumatic affection could be discovered. In the third case the pericarditis seemed to be purely idiopathic, and in the two other cases no antecedent affection was ascertained.

Although the manifestations of cerebral disorder incident to pericarditis are so varied, there are certain points of resemblance in the different cases, and, on the other hand, dissimilarity in certain respects from disorder occurring in other pathological relations. The variety of manifestations occurring in the same case, is somewhat distinctive. Different forms of delirium, coma, convulsions, &c., are developed successively during the progress of the disease. The characters pertaining to the delirium are peculiar; the patient lying in a species of coma vigil, the eyes open and fixed in one direction, not replying to questions, and incapable of being roused; this state followed by maniacal excitement, the patient shouting and apparently laboring under the fear of harm, with occasional ebullitions of hilarity. A fixed delusion of having committed some crime, appears to be a distinguishing feature. Meningeal inflammation does not give rise to this sort of delirium. Moreover, the acute pain in the head, throbbing of the carotids, injection of the eyes and face, which belong to the symptomatology of acute meningitis, are wanting. The delirium offers but a faint resemblance to that of delirium tremens, and to that which is distinguished from the latter as delirium ebriosum. It has no resemblance to the quiet, muttering delirium of continued fever, nor the active delirium which sometimes occurs in the course of febrile diseases. These points, if not diagnostic, should, at all events, excite strong suspicion of the existence of pericarditis in the absence of any symptoms pointing directly to the heart as the seat of disease. The diagnosis in such cases must rest on the presence or absence of physical signs denoting inflammation of the pericardium.

The symptoms referable to the nervous system which have been considered, are, happily, infrequent. But instances of their association with pericarditis are sufficiently numerous to show some pathological connection between the latter disease and the nervous disorder. The association is not due merely to coincidence. The instances, in fact, it is probable, are more numerous than would be inferred from the number which have been reported. As remarked by Burrows, how many have occurred in the practice of physicians who have been less candid than Abercrombie, Latham, and others, in recording their mistakes; and how great a number must have happened in the practice of those who were unable, or took no pains to distinguish these deceptive cases, it is impossible to say. It is, however, but fair to add, that some observers of large experience, who have given special attention to diseases of the heart, have not met with examples. Hope, at the time of the publication of the second edition of his work, had not observed an instance, and Stokes alludes to the subject in terms which imply that an instance has not fallen under his observation. The author last named considers the connection between the nervous disorder and pericarditis as doubtful.

Of the nature of the pathological connection assumed to exist between the nervous symptoms and pericarditis, but little is to be said. The former are not dependent on any appreciable morbid conditions of the brain or spinal cord. They are, therefore, with our present knowledge, to be regarded as functional. They have been attributed to disordered circulation, an altered state of the blood, and nervous irritation transmitted through the phrenic and pneumogastric nerves. Perhaps the most rational view is, that they proceed from the same general conditions which give rise to the associated pericarditis; in other words, that the connection is simply one of a common causation.

In addition to the symptoms which have been considered, there are none of importance, referable to the nervous system, remaining to be noticed. Pain seated in or near the præcordia, is included among the symptoms relating directly to the heart. Sleep is often more or less impaired by pain or dyspnœa. As regards the state of the mind, anxiety, apprehension, and mental depression belong to the history of the disease, and are frequently prominent symptoms.

The genito-urinary system offers no symptoms which have special relations with pericarditis. Albuminuria coexists in a certain proportion of cases, and is an important symptom of a morbid condition of the kidney, existing antecedently to the pericarditis, and upon which the development of the latter depends. Under these circumstances, it is not properly a symptom of the cardiac disease.

PHYSICAL SIGNS OF ACUTE PERICARDITIS.

In treating of the symptoms of acute pericarditis, the absence of distinctive characters derived from this portion of the clinical history of the disease has been apparent. The deficiency, as regards diagnostic points, in the symptomatic events which have been considered, enhances the importance of the physical signs. In fact, it is mainly by means of the latter that the disease may be now generally recognized with a degree of positiveness which clinical observers, not many years ago, regarded as unattainable. Of the several methods of exploration, all, save succussion, furnish signs of more or less value. I shall consider the signs obtained by percussion, auscultation, palpation, inspection, and mensuration, respectively, under separate heads, in the order in which these different methods are now enumerated.

Signs furnished by percussion.

The important signs furnished by percussion in pericarditis, are due to the accumulation of liquid effusion. The value of this method of exploration consists in the information which it affords as regards the presence or absence of liquid, the amount of distension of the pericardial sac, the variations in the quantity of liquid during the progress of the disease, and its final disappearance.

Accumulation of liquid within the pericardial sac increases the area and degree of præcordial dulness, and renders the sense of resistance in practising percussion greater than in health. Effects similar to these are produced by enlargement of the heart. The practical inquiry, therefore, arises, what circumstances distinguish the increased extent and amount of dulness due to the presence of liquid from the same effects as produced by cardiac enlargement.

The pericardium is a pyriform sac, the lower extremity forming the base. It extends upward above the base of the heart, rising as high as the cartilage of the second, and sometimes of the first rib. Unaffected by disease, it is capable of holding, as determined by Sibson's experiments already referred to, from 15 to 20 ounces of liquid. The effusion in cases of acute pericarditis rarely exceeds this amount, although the quantity is much greater in some cases of the chronic form of the disease. The area of dulness, when the sac is filled with liquid effusion, corresponds, not only to the increased size, but to the form and situation of the sac. In acute pericarditis the sac does not undergo any marked alteration in shape or dimensions. It forms, when enlarged to its full capacity, a pyriform tumor extending from the junction of the cartilage of the second or first rib with the sternum, on the left side, downward to the sixth rib or intercostal space. Laterally, in proportion to the distension, it pushes aside the lungs, increasing the space caused by the divergence of the border of the left lung, called the superficial cardiac region. If the liquid be not sufficient to distend the sac, it gravitates to the lower part, and the dilatation advances from the base upward in proportion as the accumulation goes on. After a certain amount of accumulation has taken place, the heart is raised upward, and the base of the sac somewhat depressed, so that the apex of the organ is situated at a distance from the bottom of the sac, the space below the apex being, of course, occupied by liquid.

The foregoing points, relating to the physical conditions which belong to the period of effusion in pericarditis, are involved in the circumstances distinctive of the signs furnished by percussion in this disease. The most distinctive circumstance is that already mentioned, viz., an area of præcordial dulness corresponding to the pyriform shape of the pericardial sac, commencing at the sixth costal cartilage or intercostal space, and extending upward to the second or first rib. If the sac be distended to nearly or quite its normal capacity, and no other morbid conditions are present to obscure the physical signs, its situation and shape may be delineated upon the chest with less difficulty than the space occupied by the heart in cases of enlargement, in consequence of the dulness and sense of resistance being more marked. In cardiac enlargement, the area of dulness does not present the pyriform shape which characterizes that due to pericardial effusion. It corresponds to the form and situation of the heart. The area is extended chiefly in a lateral direction below the third rib, and especially to the left of the sternum, whilst the extension is vertical rather than horizontal when the pericardial sac is distended with liquid. The distinction is marked if the accumulation of liquid be sufficient to distend the sac. It is less so, if the sac be but partially filled. The area of dulness is then widened from the base of the præcordial region, upward, to a greater or less extent, in proportion to the quantity of liquid. This lateral extension of dulness, however, is arrested, after reaching a certain distance, and, as the accumulation goes on, the dulness extends upward above the normal boundary of the base of the heart. When the pericardial sac is filled, the situation and shape of the area of dulness, as determined by percussion, are almost sufficient for the diagnosis; but if the sac be but partially filled, the signs obtained by percussion must be taken in connection with those furnished by other methods of exploration. A moderate amount of liquid will widen the area of dulness. To be appreciable, however, by percussion, when the sac is but partially filled, the præcordia must have been examined before the effusion occurred, and the fact of the area of dulness having been widened, thus determined by comparison. The degree of dulness and sense of resistance are to be taken into account. The dulness when the pericardial sac is distended, may amount nearly, or quite, to flatness, and the elasticity of the ribs is diminished in a notable manner. These effects are much more strongly marked in cases of chronic pericarditis with large effusion.

A circumstance highly distinctive of liquid accumulation in pericarditis, is the variation in the extent of dulness at different periods during the course of the disease. Effusion often taking place rapidly, if the præcordia have been examined prior to its occurrence, a remarkable increase of the area of dulness is sometimes observed after the lapse of a few hours. This enlargement may be found to have been progressive at successive examinations until it reaches a certain extent, where it may remain stationary for a greater or less period, and then decrease more or less rapidly. Fluctuations from day to day are not infrequently observed, the extent of dulness now increasing and now diminishing, until, at length, if the termination of the disease be favorable, it is reduced to the normal limits. On the other hand, the dulness from cardiac enlargement is not developed thus, as it were, under the eyes of the observer. Its extension is so gradual as, in general, to be imperceptible on comparative examinations made after intervals of weeks and even months. It never fluctuates, nor diminishes in extent. In cases of pericarditis, the daily employment of percussion is highly useful in determining, not only the existence, or otherwise, of effusion, but its increase, diminution, and final disappearance. Information concerning these points may influence considerably the treatment of the disease, as well as the prognosis.

Another distinctive circumstance is derived from the relation of the area of dulness to the point of apex-beat of the heart. In cases of cardiac enlargement, the beat of the heart is felt at the lower limit of this area. In cases of considerable pericardial effusion, the apex-beat, if it be felt, is raised above the lower border of the area of dulness from the presence of liquid. Percussion below the apex-beat yields a dull or flat sound for a certain distance, before a gastric tympanitic resonance is reached. This test is available in some cases, but not invariably. It requires that the apex-beat shall be appreciable either by the eye or touch, and the presence of gas in the stomach. The percussion should be light or superficial.

The signs furnished by percussion. in pericarditis, as already stated, relate to the effusion of liquid incident to the disease. Prior to effusion, and subsequently, this method of examination does not afford important information except in a negative point of view. The presence of lymph, it is true, increases somewhat the size of the heart, but not to an extent to give rise to an appreciable increase of the area of dulness. The availability of these signs when the pericardial effusion is abundant, may be impaired or destroyed by the coexistence of other morbid conditions affecting the adjacent parts. If the pericarditis be accompanied by pleurisy with effusion, it is difficult, or impossible to define the dulness due to a distended pericardium. Difficulty and error, were the signs furnished by percussion to be exclusively relied upon, would be likely to arise, in cases of an aneurismal sac at the arch of the aorta, or a small mediastinal tumor, or even a superabundance of fat just above the third left cartilage, all of which, in connection with enlargement of the heart, may simulate the pyramidal form of the area of dulness which is characteristic of distension of the pericardial sac. In a preceding chapter I have introduced a case of carcinoma in which the pericardial sac was filled with the carcinomatous product, giving rise to the same signs on percussion as the sac filled with liquid.[1]

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[1] *Vide* page 124.

Signs furnished by auscultation.

The auscultatory signs, in cases of pericarditis, are of great importance in their relations to diagnosis. It is more especially with respect to these, that the knowledge acquired within late years has been so useful in enabling the physician to determine, with positiveness, the existence of the disease. The signs furnished by auscultation relate, *first*, to the development of new or adventitious sounds, generally known as attrition or friction-murmur; *second*, to abnormal modifications of the heart-sounds; and, *third*, to the respiratory and vocal sounds in proximity to the præcordial region.

The sounds of attrition or friction are also called exocardial, pericardial, and peripheral sounds. The term friction-murmur is the most simple, and sufficiently expressive. The occurrence of a friction-murmur in pericarditis was first noticed by a French observer, Collin. The sound observed by him, he compared to the creaking of new leather (*bruit de cuir neuf*), and he attributed it to an unnatural dryness of the inflamed pericardium. A sound resembling the crackling of parchment was subsequently observed by Broussais. But the inauguration of friction-murmur as constituting a frequent physical sign of pericarditis, is to be dated from a publication by Stokes in 1834. The attention of Watson, of London, was directed to these sounds simultaneously with Stokes, and they were shortly afterward observed by Bouillaud, without his being aware of the prior observations of his British colaborers.

The motions of the heart in its contractions and dilatations, but more particularly its movements of rotation, involve friction of the opposed pericardial surfaces. But in the normal condition of the membrane, a friction-sound, as a rule, is not audible, exclusive of the element of impulsion in the first sound, which has been described in Chapter I of this work. This element in some persons has somewhat of a friction character; and I have occasionally discovered, in auscultating the heart, a slight rubbing or grazing sound accompanying the systole, when there were no grounds for suspecting any cardiac disease. These exceptions to the general rule do not impair the practical value of the physical sign as an indication of disease. The physical conditions necessary for the production of a morbid friction-murmur, are due to the products of inflammation deposited upon the pericardial surfaces, by which they are roughened and caused to adhere, instead of gliding smoothly and noiselessly upon each other. These products differ in different cases, and in different periods of the disease in the same case, as regards abundance, density, disposition, &c. The character of the friction-murmur is affected by these differences, but it is difficult or impossible to determine for each variety of sound a special significance, by which may be recognized, with precision, corresponding variations in the physical conditions. Like endocardial murmurs, the sounds are sometimes rough, and sometimes comparatively soft. The latter convey the idea of a gentle grazing or rubbing; the former are dstinguished as grating, scraping, or rasping sounds, and denote a more abundant and dense deposit of lymph. The creaking sound described by Collin has been attributed to the stretching of lymph which has led to partial adhesions. Walshe thinks that it may be produced when the surfaces are so closely agglutinated that attrition or separation is physically impossible, being caused by the bending and crumpling of tough, false membranes. Occasionally, according to this observer, the sounds have a clicking character. A continuous rumbling sound is sometimes heard, supposed to be due to the presence of a small quantity of liquid with which soft lymph is commingled. A churning or splashing sound occurring after a penetrating wound of the chest, causing a small aperture into the pericardium, was described to me by Dr. Knapp, of Louisville, due probably to the escape of a small quantity of blood into the pericardial sac. To attempt to describe all the varieties of friction-murmur, and apply to them different names, would render the subject needlessly complicated. It is important to know the diversities of character which they assume, only so far as this knowledge is instrumental in aiding in their recognition at the bedside.

A friction-murmur may accompany the systolic and diastolic movements of the heart, separately or combined. In the great majority of instances it accompanies both movements, or, in other words, it is double. Hence, it was described by Watson under the name " to-and-fro rubbing sound." When single, it is generally systolic. A diastolic friction-murmur occurring alone must be exceedingly rare. A double murmur may be heard in certain situations, and only a single murmur in other situations.

The intensity varies in different cases, and even at different periods in the same case, within wide limits. In some cases so faint as to be discovered only with the closest attention, it is, in other cases, sufficiently loud to be heard with the ear removed at a short distance from the stethoscope. The systolic friction-murmur may be intense, and the diastolic comparatively feeble. The converse of this must be rare. The intensity will depend, in a great measure, upon the physical conditions which give rise to the sound; but also upon the force of the heart's movements. Variation, in the latter respect, at different times in the same case, will affect the intensity of the sound. Bleeding and debilitating remedies, by weakening the heart, have been observed to lessen the intensity in a marked degree. On the other hand, the intensity is notably great when pericarditis supervenes on hypertrophy of the heart.

Under what circumstances is friction-murmur developed within the pericardium, in cases of pericarditis? In order for its production, the visceral and parietal surfaces must, of course, come into contact during the movements of the heart. The accumulation of liquid in the pericardial sac separates these surfaces to a greater or less extent, but does not necessarily prevent them from coming into contact. The movements of the heart may bring these surfaces together at certain points, and, in a recumbent position, the organ naturally gravitates to the bottom of the liquid, and rests upon the depending portion of the sac. Hence, friction-murmur is by no means uniformly arrested by an abundant liquid effusion. It is observed in some cases in which the quantity of liquid is extremely large. In many, perhaps in the majority of cases, however, it disappears during the period of effusion, probably owing to weakness of the heart's action, and not to an entire separation of the pericardial surfaces. It occurs anterior and subsequent to the period of effusion. It may be developed very soon after the commencement of the disease. Walshe states that it was detected in a case in which fatal perforation of the œsophagus and pericardium was produced in the attempt to swallow a sword, thirty minutes after the accident. Here, it was probably due to the presence of blood in the pericardial sac. I have observed it well marked six hours after the sudden occurrence of pain and other symptoms denoted an attack of pericarditis. It is rare that patients are seen at an earlier period in the disease. In that case, the disease was developed, in hospital, in connection with renal disease. It has been supposed to occur prior to the exudation of lymph, and to be dependent on dryness of the membrane. This is conjectural. Walshe states that he has known mere vascularity of a very small surface, without a particle of lymph, to produce a faint rubbing noise. This, however, is sometimes observed when the membrane may be presumed to be entirely healthy. As a rule, to which there are very few exceptions, the presence of a friction-murmur implies the exudation of lymph in more or less abundance. Moreover, as a rule, a friction-murmur is developed whenever exudation of lymph takes place and continues up to the period of effusion, if not into this period. The value of the sign in diagnosis depends, in a great measure, on this important fact. Clinical observation shows that the absence of a friction-murmur in pericarditis, if auscultation be employed with care from an early period of the disease, is a rare exception to the general rule. When not observed, assuming proper care and ability in the observer, it is probable that, in most instances, it existed prior to the case coming under observation. It may soon disappear, after becoming developed, in consequence of weakness of the heart and the presence of liquid effusion. Walshe states that he has known it to appear and disappear finally, within the space of six hours. If it disappear during the period of effusion, it often returns after the liquid is absorbed, and the pericardial surfaces again come freely into apposition. This returning friction-murmur, in conjunction with the physical signs obtained by percussion, becomes evidence of the removal of the liquid, and is, therefore, of favorable omen. It is, however, less constant at this period than during the period preceding effusion, in this respect differing from the friction-sounds incident to pleuritis, the latter being developed much oftener after, than before, the stage of effusion. The character of the sound developed in the third period of the disease may differ from that in the first period. If, prior to effusion, it was rubbing, grazing, or churning, it may become, after absorption, rasping, grating, or creaking. Adhesion of the pericardial surfaces generally arrests the sound, but this rule is not invariable. Stretching of newly-formed tissue and bending of the exudation-matter, may give rise to a creaking sound. The disappearance of the sound may be abrupt or gradual, generally the latter. Like pleural friction-murmur, it may continue for a considerable time after convalescence and apparent recovery. Walshe refers to an instance, under his observation, of its persistence for three months, continuing long after the patient's discharge from the hospital, and when he seemed to be perfectly restored to health.

Friction-murmur is to be discriminated at the bedside from endocardial murmurs. With proper care, and an acquaintance with the differential points involved in the discrimination, the instances are rare in which a practical auscultator is much embarrassed in making this discrimination. In many instances the character of the sound is sufficient to mark the distinction. The sound conveys to the mind the idea of the rubbing together of rough surfaces. This, however, is by no means to be relied upon to the exclusion of other distinctive points. A rough valvular murmur sometimes simulates closely, as far as the character of the sound is concerned, a friction-murmur. Other points are to be taken into account. A friction-murmur is generally double, that is, systolic and diastolic. This is not a distinctive point, inasmuch as a systolic and diastolic murmur exist whenever an aortic regurgitant and an aortic direct murmur are combined. Points of greater importance relate to the localization and diffusion of a friction-murmur considered relatively to endocardial murmurs. A friction-murmur is usually limited within the space occupied by the heart. It very rarely extends beyond the borders of the organ. In general, it abruptly ceases when the stethoscope is removed but a short distance without the boundaries of the heart, although it may be quite intense everywhere within the limits of the præcordia. With certain of the endocardial murmurs the rule is otherwise. They are generally heard with greater intensity at points removed from the heart, than over the heart itself. An aortic direct murmur is loudest above the base of the organ, in the second intercostal space. A mitral regurgitant murmur is usually most intense to the left of the apex. Both these murmurs are often diffused to a considerable distance from the heart, the former as far as the carotids, and the latter not infrequently over the left lateral and posterior surfaces of the chest. The maximum of intensity of a pericardial friction-murmur is generally over the body of the heart, within the superficial cardiac region. It is frequently limited to this region, or even to a portion of it, which may be either towards the base or apex, oftener the former when it is thus circumscribed. A friction-murmur does not observe rhythmical relations to the heart-sounds as do endocardial murmurs. An endocardial murmur has a certain connection with one of the sounds of the heart, and this connection is uniformly maintained. A friction-murmur, on the other hand, frequently occurs discordantly, as regards the heart-sounds, varying in rhythm irrespective of the latter. Variableness is a distinctive trait of a friction-murmur, as compared with endocardial murmurs. The latter undergo but little change with successive beats of the heart; while the former vary, even during the time occupied in an examination, as regards intensity, the occurrence of one or two sounds with a single beat, the character of the sound as regards roughness and softness, the situation in which it is loudest, &c. Its variableness, as regards duration is distinctive. Endocardial murmurs are generally persisting. The latter are rarely affected materially by change in the position of the patient. A friction-murmur is not only more intense in certain positions than in others, but it is sometimes heard only when the body is in a particular posture. Thus, it may be apparent when the patient lies on the back, and disappear when he is sitting, or *vice versâ*. Its intensity may be increased or diminished by inclining the body backwards or forwards. The explanation of this is, that in certain positions the pericardial surfaces are brought into contact at points where the physical conditions are most favorable for the production of sound. These variations in different positions are observed more especially when more or less liquid effusion is contained within the pericardial sac. A friction-murmur may be discovered by auscultating in different positions, when, were the examination limited to one position, it would fail to be apparent. Another distinctive circumstance is the apparent proximity to the ear of the auscultator. It seems to be superficially seated, resembling often the sound produced by friction of the clothing upon the stethoscope. This resemblance is sometimes so striking that the observer looks to see whether the sound be not actually thus produced. An exception to this rule is, when a friction-murmur is produced on the posterior surface of the heart. On the other hand, endocardial murmurs, as a rule, appear to emanate from points more removed from the ear, or situated deeper within the chest. This is a highly distinctive point. Finally, as indicated first by Sibson, firm pressure with the stethoscope is found generally, but more especially in young subjects, to intensify notably a friction-murmur. It does this partly by increasing the conduction, and in part by displacing the stratum of liquid, bringing the pericardial surfaces into closer apposition, and rendering the walls of the chest more resisting. In this way, a friction-murmur which has disappeared in consequence of effusion, may sometimes, according to Sibson, be reproduced. An effect of pressure is sometimes to change the character of the sound, converting it from a soft to a rough sound. Endocardial murmurs are not intensified to the same extent by pressure. It is incorrect to say that they are in no degree intensified. Their intensity is augmented in so far as the conduction is increased. It is in this way alone that pressure intensifies endocardial murmurs. This difference constitutes an important point of distinction.

Attention to the foregoing circumstances renders, in most instances, the discrimination between friction-sounds and endocardial murmurs sufficiently easy. It is, however, to be borne in mind that endocardial murmurs and a pericardial friction-murmur are frequently present in combination, pericarditis coexisting with endocarditis, or the heart being affected with valvular lesions antecedently to the development of the pericarditis. It has been supposed that the deposit of lymph at the base of the heart and about the large vessels within the pericardium, or the accumulation of a large quantity of liquid, may sometimes give rise, by pressure on the vessels, to endocardial murmur. This is doubtful. The frequent combination of the latter with the physical signs of pericarditis is to be explained by the coexistence of endocardial inflammation, or of valvular lesions. With a knowledge of the differential points involved in discriminating a friction-murmur from endocardial murmurs, the coexistence of the latter with the former does not occasion embarrassment. On the contrary, the points which are distinctive of a friction-murmur are in stronger relief when endocardial murmurs are associated. It is useful to consider that a double friction-murmur can only be confounded with either an aortic direct or a mitral systolic and an aortic regurgitant murmur existing in combination. Of endocardial murmurs, the two first of these only are systolic, and the last is the only truly diastolic endocardial murmur. Bearing these facts in mind, in cases of any doubt or difficulty, these endocardial murmurs may either be excluded, or their existence determined, by directing attention to their distinctive characters.

A pericardial murmur is to be discriminated from a murmur produced by the rubbing together of the pleural surfaces. A pleural and a pericardial friction-murmur may be combined, since pleuritis and pericarditis are not infrequently associated. The pleural murmur caused by the respiratory movements is readily distinguished from the pericardial, by the difference in rhythm. A simple expedient serves to remove any doubt as to whether it depends on the action of the heart or lungs, namely, causing the patient to suspend, for a moment, the acts of breathing; if the murmur persist, it is of cardiac origin.

But a pleural friction-murmur may be produced by the action of the heart. The friction here is *without*, instead of being *within* the pericardial sac. Addison, Stokes, and others, have reported cases exemplifying the occasional production, in cases of pleuritis, of a *cardiac pleural friction-murmur*, which may lead the auscultator into the error of supposing that pericarditis exists when there is no cardiac disease. I can bear testimony to this liability to error. In a case of pleurisy, with considerable effusion, which came under my observation in the New Orleans Charity Hospital, a well-marked, pretty intense, and rough cardiac friction-sound was apparent in the præcordia. It continued when the respiratory movements were suspended. It existed within an area from three to four inches in diameter, and was noted to extend somewhat beyond the left border of the heart. It varied in intensity with different beats of the heart, and with occasional beats was wanting. It was sometimes double and sometimes single, in the latter case being systolic. It was intensified by firm pressure with the stethoscope. It was most marked at the end of a deep inspiration. This sound continued for several days, and was listened to by a large number of physicians and students, who were accustomed to visit the ward, as an excellent specimen of a pericardial friction-murmur. Delirium tremens was developed in the case, and the patient died. I expected to find pericarditis associated with the pleurisy, but, on examination, after death, the pericardial sac contained a moderate quantity of transparent serum, and the membrane was perfectly healthy. The heart was in all respects normal; the endocardium presented a natural appearance, and no valvular lesions existed. The left pleural sac contained a large quantity of turbid serum. The lung extended downward, in front to the level of the nipple. It was connected by tender adhesions to the outer surface of the pericardium, and the costal and pleural surfaces were also united by tender adhesions. The friction-murmur was evidently produced either by the rubbing together of the outer surface of the fibrous sac inclosing the heart against the adjacent lung (which overlaid the heart), or of the opposed pleural surfaces above the heart. In view of this case, I am prepared to concur in the opinion of Addison, that "auscultation will not always enable us to distinguish a friction-sound produced *within*, from a friction-sound produced *without* the pericardial sac."[1] Walshe gives the following as the circumstances which argue in favor of a friction-murmur of cardiac rhythm being of pleural, and not of pericardial, origin: "The limitation of the sound to either edge, generally the left, of the cardiac region; fixity in one or more particular spots; cessation complete, or, what is more common, occasional, with certain beats of the heart, when the breath is held; and marked unsteadiness in the intensity and quality of the friction-sound." These circumstances were not fully available for the discrimination in the case which I have cited. It may be added to the account of the case that, in making clinical remarks, attention was called to the occasional occurrence of a cardiac pleural friction-murmur, but I confess that I did not regard the case as furnishing an illustration till the chest was examined after death. The sound was not limited to the edge of the cardiac region, but was heard over an area of from three to four inches in diameter. It extended, however, beyond the left border of the heart. It did not cease when the breath was held, but it was most marked at the end of a full inspiration, while a pericardial friction-murmur is usually most marked at the end of the act of expiration. "Unsteadiness in the intensity and quality of the friction-sound" certainly characterizes a true pericardial friction-sound in certain cases.

In conclusion, a true pericardial friction-sound, as regards diagnostic significance, is, perhaps, the most definite and reliable of all the signs obtained by physical exploration of the chest. Exclusive of cases in which the pericardium is perforated, so that a small quantity of blood escapes into the sac (which is not necessarily followed by inflammation), it is pathognomonic of pericarditis.

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[1] Guy's Hospital Reports, vol. iv. From Bellingham, op. cit.

Since the publication of the first edition of this work, I have met with two cases in which a cardiac friction-murmur produced outside of the pericardial sac, was recognized as such, during life, and verified by examinations after death. In one of these cases the patient had pneumonia affecting the whole of the left lung. A friction-murmur with cardiac rhythm, continuing when the respirations were suspended, was considered as produced outside of the pericardial sac, because there was no evidence of pericardial effusion, and the murmur was limited to the left border of the heart. The autopsy showed no evidence of pericarditis, and the pleural exudation was so situated that the production of the murmur outside of the pericardium was intelligible. The immediate cause of death in this case was the formation of a heart-clot in the right cavities of the heart. The following account of the second case was noted at the time of its occurrence. A patient, aged about 45, was admitted into Bellevue Hospital ten days before his death. He was much prostrated, and presented the signs of solidification of lung over the middle third of the chest on the left side in front and behind. The expectoration was fetid, and pulmonary gangrene was suspected. He failed progressively, and lay in a semi-somnolent state, but complaining of no pain in the head. The day before his death my attention was called to a cardiac murmur which had not before been discovered. The murmur was evidently due to friction; it was double, superficial, and intensified by pressure. It served to illustrate a cardiac friction-murmur to one of my private classes in auscultation. It was limited to a small space, on the left of the sternum, between the second and fourth ribs, the space in this situation being on the confines of the præcordia. There was no evidence of liquid in the pericardial sac. In view of the situation and limitation of the murmur, the existence of pulmonary disease in that situation, and the absence of pericardial effusion, I predicted that the murmur would be found to be a cardiac pleural friction-murmur. The autopsy showed that at the situation where the murmur was heard, there was a pulmonary cavity. The pleura which formed a portion of the wall of this cavity was adherent to the pericardial sac, and the lungs were adherent to the thoracic walls at this point. The lungs were not tuberculous, and the cavity was doubtless due to circumscribed gangrene. The inner surfaces of the cavity were in apposition over a space of the size of half a dollar. The relation of the walls of the cavity were such as to render it evident that the murmur was produced by the rubbing together of its inner surfaces. There was no evidence of pericarditis, and the heart was free from valvular lesions. Examination of the head revealed the existence of chronic meningitis. This case is interesting from the fact that the cardiac friction-murmur, although produced outside of the pericardial sac, was not a cardiac pleural friction-murmur, being produced within the pulmonary cavity. In this respect the case is perhaps unique.

The heart-sounds undergo certain abnormal modifications in pericarditis. Early in the disease, prior to liquid effusion, either they are not materially affected, or they are intensified by the excited action of the heart. An abundant accumulation of liquid occasions marked modifications, more especially of the first or systolic sound. The impulsion of the apex against the thoracic walls being either prevented or greatly weakened, the element of the first sound, which has been designated the element of impulsion, is impaired or lost; the valvular element is left isolated, or it becomes predominant. Hence, the first sound is enfeebled and shortened, resembling the second sound in quality and duration. The second sound is affected only as regards intensity, and, in this respect, less than the first sound. The second sound over the whole præcordia is the accentuated sound; it is sometimes the only sound discoverable, the first sound being suppressed. The muscular weakness of the heart, arising from compression and the paralyzing influence of the pericardial inflammation, favors these effects. Both sounds appear to be further removed from the ear than in health. This sense of distance is most marked when the patient lies on the back, because the heart is then actually further removed from the anterior walls of the chest. The apparent distance diminishes perceptibly when the sitting posture is assumed, and still more when the patient leans forward. After the absorption of the liquid, the normal characters of the sounds return, except so far as they may be modified by weakness of the heart. In these remarks, it is assumed that valvular lesions are not present. These will, of course, be likely to affect the sounds of the heart, irrespective of the pericarditis.

Auscultation of the respiration and voice may be employed, in conjunction with percussion, in determining the space occupied by the pericardial sac when distended with liquid. The limits to which the respiratory murmur extends in a direction toward the præcordia in a measure serve to define the boundary of this space; if these limits coincide with the dulness on percussion, the two methods mutually confirm each other, as regards the accuracy of their respective results. But vocal resonance is more available for this purpose than the respiratory murmur. It is found to cease abruptly where dulness or flatness gives place to vesicular resonance on percussion. These different classes of signs, thus, concur in delineating the line of demarcation between the lungs and pericardium. This application of auscultation presupposes that the lungs are free from disease. Auscultation of the respiration and voice is obviously important in determining the nature and extent of pulmonary affections which may be associated with pericarditis.

Signs furnished by palpation.

These are important in the diagnosis of pericarditis. Prior to the stage of effusion, the beating of the heart may be found, on palpation, to be abnormally forcible, violent, diffused over a larger area than in health, and sometimes tumultuous. The action of the heart, as thus ascertained, may be in striking contrast with the pulse. Hope enjoined the rule of habitually placing the hand upon the præcordia in all cases of disease, in order that attention may be directed to the heart in instances in which, from the absence of obvious symptoms referable to that organ, cardiac disease is liable to be overlooked. By observing this rule, pericarditis may sometimes be discovered earlier than would otherwise be the case. A practical object in examining the præcordia, by palpation, prior to effusion, is enforced by Walshe, viz., to determine the situation of the apex-beat with reference to subsequent comparisons. When effusion takes place, the apex of the heart is raised and carried to the left. The point of apex-beat which, prior to effusion, was situated in the fifth intercostal space, may be found, after effusion has occurred, to be raised to the fourth intercostal space and carried to the vertical line of the nipple, or, possibly beyond this line. This change, taken in connection with other signs, is significant of pericardial effusion; but it must be recollected that causes other than pericardial effusion alter, in the same way, the point of apex-beat, for example, tympanitic distension of the stomach. An abundant accumulation of liquid suppresses all impulse. When the beat is found to disappear, after the presence of liquid has been already determined, an increased accumulation is to be inferred; afterward, as absorption of the liquid goes on, the heart's impulse becomes again appreciable, at first elevated above its normal position, and it may, or it may not, subsequently fall to the point where it was felt prior to the effusion of liquid. It is to be borne in mind that various morbid conditions, other than pericardial effusion, may cause suppression of the apex-beat; such as pleuritic effusion, pulmonary emphysema, dilatation of the heart, fatty degeneration, &c. Excluding these, the following series of events, occurring successively in connection with other signs denoting pericarditis, become highly diagnostic: increased force of the apex-beat, followed by diminished force, the point where it is felt raised and carried to the left, suppression of the beat, and its subsequent reappearance at or somewhat above the point where it is felt in health.

A physical sign determined by palpation is a sensible vibration of the thoracic walls in the præcordial region, caused by friction of the pericardial surfaces roughened by an abundant deposit of dense lymph. The sensation communicated to the hand is analogous to that which accompanies a friction-murmur due to the respiratory movements in some cases of pleuritis. This sign, first described by Stokes, is called the *pericardial friction-fremitus.* It is produced by the same physical conditions which give rise to friction-murmur, and it is always accompanied by the latter. It occurs, however, only in a certain proportion of the cases in which a friction-murmur is heard, and it rarely continues as long as the latter. It requires a greater degree of roughness of the pericardial surfaces than always obtains in pericarditis, and, also, a certain amount of vigor in the heart's movements. It occurs prior to the period of effusion, but very rarely after this period, the movements of the heart, at this stage of the disease, not being sufficiently strong to produce it. It is not incompatible with a small amount of liquid effusion, but it invariably ceases when the quantity of liquid is large. It is not to be confounded with the purring thrill which occurs in connection with valvular lesions. It is also to be discriminated from pleural tactile fremitus. The latter being produced by the respiratory movements, ceases when respiration is suspended; while pericardial fremitus, being produced by the movements of the heart, is not arrested by holding the breath. The sensation of rubbing, or friction, distinguishes it from purring thrill. As a physical sign, the significance of tactile pericardial fremitus is neither more nor less than that of a rough friction-murmur.

It is stated by Walshe that when the pericardial sac contains a certain quantity of liquid, the heart's impulse is sometimes felt after the systolic sound is perceived by the ear, a distinct interval separating these events which in health occur synchronously.

Signs furnished by inspection.

During the stage of liquid effusion, if the pericardial sac be considerably distended, an unnatural prominence or arching of the præcordial region is sometimes apparent to the eye, especially if the subject be young. This sign of pericardial effusion was first pointed out by Louis. It is less frequent and marked in cases of acute pericarditis, than when the disease assumes a chronic form, with a very large accumulation of liquid. The effects of great distension of the pericardium, as determined by the different methods of exploration, will be noticed under the head of chronic pericarditis. Whenever the accumulation is sufficient to occasion an obvious projection of the præcordia, the intercostal spaces are, at the same time, widened and raised to the level of the ribs. These appearances are limited to a space on the chest corresponding to that which the distended pericardium occupies; and the prominence may present, indistinctly, an outline of the pyriform shape of the pericardial sac. Under these circumstances, the impulse of the heart is rarely either seen or felt, a fact which serves to distinguish prominence of the præcordia produced by pericardial effusion, from that due to enlargement of the heart. In the latter case, one or more points of impulse are usually both seen and felt.

If the quantity of liquid be sufficient to produce visible enlargement of the præcordia, the respiratory movements on the left side are somewhat restrained. A disparity between the two sides, in this respect, may be obvious. The respiratory movements of the left side may also be restrained prior to the occurrence of effusion, in consequence of the pain felt during inspiration. Deficient respiratory motion is not, therefore, alone a sign that effusion has taken place. According to Walshe, if the quantity of effusion be moderate, the costal expansion of the left side may even be greater than in health, in compensation for some depression of the diaphragm.

Inspection is sometimes important as a means of determining the situation of the apex-beat of the heart in cases in which this may be seen and not felt. Not infrequently the motion occasioned by the beat may be discovered by the eye, when it cannot be appreciated by the touch.

Undulatory movements in the intercostal spaces over the pericardium distended with liquid are occasionally observed, due to motion produced by the heart's action. This sign possesses small intrinsic value, *first*, because of its infrequency, and *second*, because it is not easy to distinguish it from the movements styled by Walshe *quasi undulatory*, caused by the motions of the heart itself when much dilated and in contact with the thoracic parietes over an enlarged area. The latter I have often observed, while it has not occurred to me to witness true undulation from liquid. Occurring in concurrence with other signs denoting unequivocally pericardial effusion, it has a positive significance.

If the quantity of liquid effusion have been sufficient to occasion a visible prominence of the præcordia, the absorption of the liquid may be followed by an obvious præcordial depression. This is sometimes marked, and is important as one of the signs of ancient pericarditis.

Signs furnished by mensuration.

When the præcordia is rendered abnormally prominent by distension of the pericardial sac, during the period of effusion, this fact may be determined by mensuration. Diametrical measurement, with callipers, is best adapted to this object, for the same reasons as in cases of prominence due to cardiac enlargement. The normal deviations from equality of the two sides of the chest, as regards the results of diametrical measurement, are to be borne in mind.[1] The existence of undue præcordial prominence is determined by the eye sufficiently for practical purposes; but the callipers may be employed in confirmation of the evidence afforded by inspection. Owing to the rapidity with which, in certain cases, the liquid, on the one hand, accumulates, and, on the other hand, diminishes, præcordial prominence may be suddenly produced or increased, and as suddenly diminish or disappear. These variations may be ascertained with greater precision by mensuration than by the eye, and in recording cases which may be reported, it is more satisfactory to note the results of measurement, in addition to the appearances presented on inspection.

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[1] *Vide* Chapter I, page 69.

SUMMARY OF THE PHYSICAL SIGNS OF ACUTE PERICARDITIS.

Percussion.—Enlarged area of præcordial dulness; the extent of this area greater in a vertical than in a transverse direction; its shape corresponding to the pyramidal form of the pericardial sac when distended; the dulness within this area, and the sense of resistance on percussion greater than over the præcordial region in health, or in cases of enlargement of the heart. These signs denote an abundant effusion within the pericardial sac. Moderate or small effusion denoted by increased width of the area of dulness at the lower and middle portions of the præcordial region. The increase of the area of dulness taking place within a few days or hours, and progressing rapidly; its extent varying on different days during the course of the disease. Dulness from the presence of liquid below the point of the apex-beat of the heart. Diminution of the area of dulness, with more or less rapidity, in the progress of the disease toward convalescence, and its final reduction to its normal limits when convalescence is established.

Auscultation.—A friction-sound developed, usually, soon after the commencement of the inflammation, depending on the exudation of lymph; rarely wanting during the period of the disease which precedes that of liquid effusion; frequently, not invariably, disappearing during the period of effusion; often returning after the absorption of liquid, and sometimes persisting after adhesion of the pericardial surfaces has taken place. Intensification of the heart-sounds at the commencement of the disease, or prior to liquid effusion; during the period of effusion, both sounds weakened, but especially the first sound; the element of impulsion in the first sound notably impaired or lost, and this sound, therefore, consisting of the valvular element alone, resembling the second sound as regards quality and duration; the sounds apparently distant, and the apparent distance greater when the patient is recumbent on the back. Cessation of respiratory murmur and vocal resonance, concurring with the results of percussion, in determining the enlarged area of præcordial dulness dependent on distension of the pericardial sac.

Palpation.—Prior to the period of effusion, the cardiac impulse abnormally forcible, violent, extending over a larger space than in health, and sometimes tumultuous beating of the heart. After effusion, the point of apex-beat raised and carried to the left of its normal position. Suppression of the apex-beat, if the quantity of liquid be large. Return of the beat when the liquid diminishes. Vibration of the thoracic walls in the præcordia before, and sometimes after, the period of effusion, constituting tactile friction-fremitus. Retardation of the apex-beat in some cases, after a certain amount of effusion, so that the first sound precedes it by a distinct interval.

Inspection.—Prominence or arching of the præcordial region in some cases during the period of effusion, if the pericardial sac be distended, observed chiefly in young subjects; the prominence presenting an indistinct outline of the pyriform shape of the pericardial sac. Restraint of the respiratory movements of the left side, if the quantity of liquid be large, and, also, prior to effusion, in some cases, from pain felt in the act of inspiration. Undulatory movements in the intercostal spaces over the pericardium distended with liquid, in a very small proportion of cases. Depression of the præcordial region in some cases, after the absorption of liquid.

Mensuration.—Prominence of the præcordia, in some cases, produced by liquid accumulation in the pericardial sac, determined by callipers. Sudden development or increase of prominence, and its sudden or rapid disappearance.

DIAGNOSIS OF ACUTE PERICARDITIS.

The diagnosis of pericarditis formerly was confessedly difficult in all cases, and often impossible. So long as the discrimination rested mainly on symptoms, it could rarely be made with positiveness. Laennec candidly acknowledged that the disease was not to be recognized, but its existence only conjectured. It was seen while passing in review the symptomatic phenomena that none of these are distinctive. As regards symptoms pointing to the heart, the disease is not infrequently absolutely latent. Moreover, in a certain proportion of cases, it is associated with other affections which, as it were, drown its manifestations. The disease now, as heretofore, is very rarely, if ever, ascertained to exist with certainty by those who rely in diagnosis on symptoms alone. And since physical exploration is still neglected to much extent, pericarditis is habitually overlooked by not a very few medical practitioners. With the aid of physical signs, the diagnosis may generally be made with ease. These have been sufficiently considered, but there are certain sources of inadvertency and embarrassment which, in order to be avoided, should be clearly understood and impressed upon the mind.

The disease is liable to pass undetected because its existence is not suspected and attention is not directed to the condition of the heart. It is important to bear in mind the pathological relations of the disease, in order to be prepared to expect it, and to be on the watch for the earliest evidence of its development. During the progress of acute rheumatism, the præcordial region should be daily explored with reference to the signs of pericarditis, as well as those of endocarditis. By so doing, a friction-murmur may sometimes be discovered when the patient makes no complaint of pain or other symptoms denoting that the pericardium has become involved. So, when a patient is known to be affected with disease of the kidneys, the fact that inflammation of the pericardium, as well as of other serous structures, is apt to be developed, is not to be forgotten, and examinations of the chest should not be neglected. In the eruptive and continued fevers, attention should be directed to the heart, and, indeed, it is well to adopt the practical rule enforced by Hope, which has been already mentioned, namely, to employ at least palpation habitually in all cases of disease.

The coexistence of either pleurisy or pneumonia, is liable to lead the practitioner to overlook pericarditis, *first*, because, having ascertained the existence of these affections, he may attribute all the symptoms to them, and not carry his inquiries farther; and, *second*, because these affections obscure the symptoms, and, to some extent, the signs of pericardial inflammation. The diagnosis of the latter is, in fact, sometimes difficult under these circumstances. The heart should be interrogated as far as possible. A pericardial friction-murmur may be discovered even when the organ is displaced to the right of the sternum. Symptoms referable to respiration and the circulation, when they are out of proportion to the pulmonary affection, should excite strong suspicion of cardiac disease. Liquid effusion in the pericardium when, at the same time, there is an abundant effusion in the left pleural sac, is by no means easily determined; but, with due attention, the pericardial accumulation may sometimes be disconnected from the pleural by prominence of the præcordia, the vertical extent of præcordial dulness, &c. An abundant effusion into the right pleural sac does not interfere materially with the signs of pericardial effusion, and the diagnosis of the latter may be made with positiveness. The occasional occurrence of a cardiac friction-murmur produced outside of the pericardial sac is to be recollected especially in connection with the subject of pericarditis associated with pleurisy seated in the left side of the chest. This sign is a source of fallacy, and this fact should lead the practitioner not to commit his mind too unqualifiedly to a diagnosis based exclusively on the existence of a friction-murmur.

I have known acute pericarditis, disconnected from any other thoracic affection, to be considered and treated throughout the disease as pleurisy; but the diagnosis was based on symptoms alone. A tolerable knowledge of physical exploration enables the diagnostician to exclude, on the one hand, and, on the other hand, to ascertain the existence of pleurisy and pneumonia. If the examination of the chest be limited to the anterior surface, the physical signs of liquid within the pericardial sac might be attributed to pleural effusion in the left side. A proper examination of the whole chest obviates liability to this error. The signs of effusion are limited to the anterior surface. Percussion and auscultation show the presence of lung in the lower posterior portion of the chest; and it is precisely in the latter situation that the signs of pleuritic effusion are first manifested and most marked. This error is excusable on no other ground than inability to employ the means of arriving at a correct diagnosis. The occasional occurrence of symptoms referable to the brain and spinal cord, in connection with pericarditis is to be borne in mind. These symptoms may mask completely those pertaining to the cardiac affection, simulating various affections of the nervous system, namely, mania, apoplexy, tetanus, &c. The peculiar characters which serve to distinguish these cases are to be kept in view, and careful attention directed to the heart in all instances in which these affections appear to be present.

Pericardial effusion, occurring without inflammation, has not yet been referred to. Dropsy of the pericardium, or hydro-pericardium, rarely occurs to an extent sufficient to occasion great distension of the sac. It occurs very rarely, if ever, except in conjunction with effusion into other serous cavities, and the areolar tissue, constituting general dropsy. The effusion into the pleural cavities is proportionately greater than into the pericardial cavity. The physical signs of a certain quantity of purely serous or dropsical effusion are, of course, the same as when the effusion is combined with lymph, or, in other words, inflammatory. The pericardial sac, in proportion to its distension, occupies the same space, enlarges the area of dulness to the same extent, the latter presenting the same pyriform shape, &c., in the two cases. The discrimination, however, rarely offers much real difficulty. Liquid effusion in pericarditis is generally preceded and accompanied by more or less of the symptoms pointing to the latter affection, such as pain, tenderness, febrile movement, &c. It is preceded almost invariably, and not infrequently accompanied, by a cardiac friction-murmur. If pleuritic effusion be also present, as determined by physical signs, it is not hydrothorax, but is due to coexisting inflammation of the pleura; and the inflammatory pleuritic effusion is generally limited to one side. On the other hand, hydropericardium is an element of general dropsy; œdema or anasarca, hydroperitoneum, and hydrothorax, are at the same time present. A friction-sound is never developed. In the vast majority of cases, the patient is affected with either renal disease or organic disease of heart, and both affections may be united. With due attention to these differential points, the two kinds of effusion, viz., dropsical and inflammatory, need not be confounded.

In leaving the subject of diagnosis, the great importance of becoming familiar and practically conversant with the physical signs is to be impressed. With this knowledge, the practitioner will rarely be long at a loss in determining whether acute pericarditis be, or be not, present.

PROGNOSIS IN ACUTE PERICARDITIS.

Acute pericarditis is never a trivial, and is often a formidable affection. The fatality, however, is due, not so much to the disease itself, as to the condition of the system, the pathological relations of the disease, and coexisting affections. Cases of idiopathic pericarditis are so rare that statistical data for determining the rate of mortality are, as yet, wanting. Of 106 cases of pericarditis variously complicated, which were analyzed by Louis, 36 died. The reports of different observers, as regards the proportion of fatal cases in their own experience, differ considerably; and this would be expected, in view of the great variation in the tendency to a fatal result according to the different circumstances under which the disease is developed. Hope, whose opportunities for observation must have been extensive, makes the remarkable statement that, in ten years, he had not lost a patient with acute pericarditis, ascribing this success to the treatment pursued. Yet, according to the observations of others, the disease is almost invariably fatal when developed in certain pathological connections.

In the majority of cases, pericarditis occurs in connection with acute articular rheumatism. Occurring in this connection it rarely proves fatal. In 84 cases reported by Latham, McLeod, and Bouillaud, there were but 8 deaths; and, in more or less of these fatal cases, endocarditis coexisted. Rheumatic pericarditis, thus, may be expected to end in recovery, or, at least, not to terminate fatally as an acute affection. It is otherwise when the affection is developed in connection with renal disease; a fatal result occurs in the larger proportion of cases. Death takes place in a large proportion of the cases in which it is associated with pleurisy or pneumonia. The proportion of fatal cases of pericarditis developed in connection with the eruptive and continued fevers, pyæmia, &c., must be immensely large; but statistical data are wanting to determine the ratio with any approximation to exactness. The disease is generally fatal when associated with marked disorder of the nervous system, giving rise to mania, tetanus, chorea, &c.

As regards the fatality in my own experience, the following are the results of an analysis of 19 recorded cases, with reference to this subject. Of the 19 cases, death occurred in 12, and in 7 the disease ended in recovery. Of the 12 fatal cases, renal disease was ascertained to exist in 3; pleurisy existed in 5; pneumonia in 2; tuberculosis of the lungs in 3; maniacal delirium in 2; and in 1 case no important complication was ascertained. In the case last mentioned, the disease did not present any alarming symptoms, but death occurred suddenly, apparently from syncope, while the patient was at stool. The pericardial sac contained a pint of turbid serum. The heart presented two patches of lymph, one of the size of a dollar, and the other of the size of a shilling piece. The pulmonary organs were free from recent disease, but old pleuritic adhesions existed on both sides, and they were universal on the right side. The chest was alone examined. Of the 7 cases ending in recovery, in 4 the disease was developed in connection with rheumatism; in 1 case it was apparently idiopathic, and was associated with maniacal delirium; in 1 case pneumonia coexisted; and in 1 case it followed albuminuria, succeeding scarlatina.[1]

Of 31 cases analyzed in revising this chapter for the second edition, death occurred in 15, and 16 cases ended in recovery. Of the 15 fatal cases, renal disease was ascertained to exist in 3, the kidneys in some cases not having been examined. Pneumonia existed, prior to the pericarditis, in 7. In one of these 7 cases, the kidneys were ascertained to be diseased, and in 1 case gangrene of lung took place. The pneumonia affected the whole of one lung in 5 cases, and in 1 case it was double. Valvular lesions and enlargement of the heart existed in 1 case. Emphysema and hepatic abscess existed in 1 case. In 1 case there were polypoid tumors in the right auricle, and an occlusion of one of the primary branches of the pulmonary arteries, from an embolus, the patient in this case having been found dead. In 2 cases only was the disease rheumatic pericarditis. In these 2 cases no other disease than the rheumatism and the pericarditis was ascertained during life, but there was no post-mortem examination in either case, and death in 1 case occurred the day after admission into hospital, the pericarditis having existed for a week. Of the 16 cases ending in recovery, the disease was idiopathic and uncomplicated in 1 case. It was traumatic, produced by a penetrating wound with a fork in 1 case. It was associated with acute rheumatism and pleurisy in 1 case. It was associated with pleurisy, without rheumatism in 1 case. It was associated with double pneumonia and rheumatism in 1 case, and with pneumonia affecting a single lobe and rheumatism in 1 case. Pleurisy without rheumatism coexisted in 2 cases. In 8 cases rheumatism existed without any other complication than the pericarditis.

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[1] It is proper to state that the foregoing collection of cases does not include all that have fallen under my observation, but only those of which I find notes among my clinical records. It probably embraces the greater proportion of fatal cases which I have observed, while, of a considerable number of cases of rheumatic pericarditis not ending fatally, I have not preserved notes.—*Note to first edition.*

The foregoing note is still more applicable to the ten years which have elapsed since the publication of the first edition of this work.—*Note to second edition.*

The duration of the disease is variable. It may prove rapidly fatal. In a case reported by Andral, death occurred in twenty-seven hours; but it is extremely rare that it runs with this rapidity to a fatal issue. It continues usually from one to two weeks. If it do not prove fatal within this period, it ends either in recovery or in the chronic form of the disease. The latter will presently be considered under a distinct head.

The termination, in favorable cases, is in more or less adhesion of the pericardial surfaces. It may fairly be doubted whether the inflammatory products are ever completely removed by absorption, leaving the surfaces of the membrane unattached and presenting no traces of the disease. Some, however, have contended that this complete resolution occasionally takes place. As a rule, certainly, permanent effects are left here, as after recovery from inflammation affecting other serous structures, consisting of adhesions by means of newly organized tissue which becomes more and more firm with age. Adhesion may take place over the entire surfaces of the membrane, the pericardial sac being obliterated, like that of the tunica vaginalis after the radical cure of hydrocele. In fact this is the rule. Of 70 cases of pericardial adhesions analyzed by Louis, they were general in 60, and partial in 10; and of 86 cases of old adhesions analyzed by Chambers, 51 were universal, 4 nearly so, and 29 partial.[1] The subject of pericardial adhesions, with reference to their remote effects upon the heart, and the diagnosis, will be noticed after chronic pericarditis has been considered. The most favorable termination of acute pericarditis, next to complete resolution (the occurrence of which is doubtful), is the formation of circumscribed white patches, consisting of thin layers of dense lymph, firmly agglutinated to the membrane, becoming nearly as smooth and polished as the membrane itself. These white patches (*maculæ albidæ*), as an effect of circumscribed or partial pericarditis, have been already noticed. It is possible that patches similar to these are, in some cases, the only permanent effects of acute, general pericarditis; but this must be considered as doubtful.

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[1] Decennium Pathologicum. *Vide* Bellingham, op. cit., part ii, p. 309.

The mode of dying, in cases of acute pericarditis, is not uniform. When life is destroyed by the disease *per se*, the result is immediately due either to sudden syncope or gradual asthenia. The arrest of the circulation is the immediate cause of death; and this is owing to paralysis of the heart from the combined influence of the mechanical pressure of liquid effusion and the proximity of the inflamed membrane to the muscular fibres of the organ. In the latter respect, the influence is analogous to that of inflammation of the peritoneum on the muscular coat of the intestines. It is important to keep in view this twofold influence in producing a tendency to a fatal result, since it should govern, to a considerable extent, the indications for treatment. But in the great majority of fatal cases of pericarditis, concomitant affections contribute, in no small degree, to the fatal result, and the mode of dying will, in a measure, be determined by these. For example, when the disease is associated with pleurisy or pneumonia, apnœa is involved as an immediate cause of death. Again, when developed in the course of renal disease, the powers of the system being exhausted by the latter, the tendency to death may be by slow asthenia; or coma and convulsions may be induced as effects of uræmia. Coma precedes death in the cases in which pericarditis simulates various affections of the brain and spinal cord. A liability to sudden death from syncope, during the period of effusion, must not be overlooked. This may occur after some unusual muscular exertion, or a sudden change from a horizontal to a vertical position. An instance in which death occurred suddenly and quite unexpectedly, in a case under my observation, has been referred to, fatal syncope being induced by getting out of bed and going to stool. The inflammation in this case, as shown by the appearances after death, as well as the symptoms during life, was not intense, and the pericardial sac did not contain more than a pint of liquid effusion.

TREATMENT OF ACUTE PERICARDITIS.

In the treatment of most acute inflammations, the general symptoms, the condition of the system, the pathological relations of the disease, &c., are more immediately involved in therapeutical indications, than the local processes which constitute the inflammatory affection. The treatment of acute pericarditis does not form an exception to the rule embodied in this statement. Acute pericarditis is developed under circumstances so widely different in different cases, that, assuming the local characters of the inflammation to be identical, the indications for treatment are by no means uniform. Measures useful in some cases are pernicious in other cases. Methods of management diametrically opposite are indicated by different circumstances connected with the disease. It follows that the treatment cannot be reduced to a fixed formula applicable to all cases. Here, as in other affections, the significant saying of Chomel is pertinent, viz., the disease is not to be treated so much as the patient affected with the disease. In the majority of cases, pericarditis occurs in connection with acute rheumatism, and in a certain proportion of cases it is developed in the course of Bright's diseases. The local inflammation, in all essential points, so far as these are appreciable, may be the same in these two groups of cases, but in other respects, the difference is very great. Rheumatic pericarditis involves small immediate danger to life, although its remote evils may be serious. In renal pericarditis (if this expression may be used) the immediate danger to life is imminent. The chances of recovery in the latter are less than the chances of death in the former. Both forms of the disease are dependent on special diathetic or constitutional conditions which are essentially dissimilar. The active rheumatic diathesis is acute, transient in duration, affecting the young; the uræmic condition is incident to a chronic, persistent affection, and occurs at a later period in life. The ability to bear up under any grave local disease, and to support potent remedial agencies, is as different as are the pathological relations of the disease in these two forms. In addition to these, a variety of modifying circumstances are present in different cases of pericarditis, not peculiar to this disease, but affecting the symptoms, the powers of the system, &c., so as to influence, in various and opposite modes, the leading objects of treatment. It is not to be inferred from these remarks that, in the treatment of pericarditis, importance does not belong to the local morbid conditions. It is by means of the vital and mechanical effects of the latter that the disease proves destructive to life. The objects of treatment relate to the local morbid conditions; but in promoting these objects, the indications are in a great measure derived from the circumstances just referred to.

It is obvious that the first and most important object of treatment, were it attainable, would be an arrest of the inflammation. But we are not warranted in assuming this as an object to be effected by therapeutical measures. With our present knowledge, we cannot say that certain methods of management will cut short the inflammatory processes here, more than in other situations. Potent means adopted for that end, not only prove ineffectual, but involve the risk of doing harm rather than good. To abridge the duration of the inflammation is an object, the importance of which is sufficiently obvious. How far this object is attainable must be considered as doubtful, but we are perhaps justified in regarding it as an end to which therapeutical measures are to be directed. To endeavor to diminish the intensity of the inflammation, is a legitimate object of treatment. The products of inflammation, solid and liquid, in this situation, involve serious evils and danger. It is an object of treatment to endeavor to lessen these, and to promote their absorption. To aid in maintaining the vigor of the heart under the effects of the disease is an important end to be kept in view in the treatment. I shall proceed to notice the more important of the therapeutical measures which are supposed to promote the objects or ends of treatment in acute pericarditis. These may be arranged under the following heads: Bloodletting, mercurialization, sedatives, revulsives or counter-irritants, opium, stimulants, and eliminatives.

Bloodletting.—The two authors whose labors have contributed most to the recent progress made in the knowledge of diseases of the heart, viz., Bouillaud and Hope, both advocate strongly the importance of bloodletting in cases of acute pericarditis. Bouillaud employs, in this disease, his methods of copious bleedings, repeated once or oftener daily for four or five successive days (*coup sur coup*), with which medical readers are familiar.

Hope's method differs from that of Bouillaud in the employment of bloodletting only at the outset, and carrying it to the extent of producing a prompt and decided impression on the heart's action. Each advocates the abstraction of blood both by venesection, and locally, by leeching or cupping. Writers of a more recent date (Stokes, Todd, and others) distrust the efficacy of this remedy, and attribute to it in many cases unfavorable effects. Without entering into a discussion of the subject of bloodletting, which would here be out of place, it is sufficient to say that, with certain qualifications, the general principles which should regulate the employment of this potent remedy in other acute inflammations, are applicable to the treatment of acute pericarditis. Its indiscriminate use in this, as in other inflammations, cannot but be productive of much harm, whereas, judiciously employed, it may in certain cases do good. The practical questions are, under what circumstances is bloodletting indicated, and what are the contraindications to its use? A person in fair health and vigor, attacked with acute pericarditis, as an idiopathic or a rheumatic affection, may be a proper subject for bloodletting at the onset of the disease. Resorted to under these circumstances, it will not cut short the disease, and perhaps not abridge its duration; but it may contribute to diminish the intensity of the inflammation, and thus, without risk of injury, not only afford immediate relief, but lessen the evils and the danger, proximate and remote, which are involved in the disease. The amount of blood to be abstracted, must be determined by the constitution, habits, &c., of the patient, the symptoms referable to the circulation, and the immediate effects upon the vascular system. Whether the bloodletting shall be general or local, or both, is to be determined mainly by the quantity of blood which it is deemed desirable to withdraw, and the comparative convenience of venesection and cupping or leeching. It is difficult to conceive of any important difference between these different methods, as regards their effect on the disease, except so far as concerns the rapidity with which the blood is removed, and the amount abstracted. The benefit derived from bloodletting will be evidenced by relief of pain, greater freedom of breathing, diminished force and greater regularity of the heart's action. These, then, are the circumstances which may indicate bloodletting, viz., the disease idiopathic, or rheumatic; occurring in a patient previously healthy and tolerably vigorous; the inflammation recently developed, or, in other words, the disease being in its first period, and, to these is to be added, a certain degree of intensity or acuteness of the inflammation, as manifested by pain, development of the pulse, &c. The indications based on these circumstances are present in a certain proportion of cases of pericarditis. The contraindications, however, are present in much the larger proportion of cases. Pericarditis occurring in connection with Bright's disease, rarely, if ever, calls for bloodletting. The anæmic condition incident to that disease, constitutes a contraindication. Anæmia from other causes, weakness or deterioration of the constitution from previous or coexisting disease, habits of intemperance, &c., are contraindicating circumstances. Bloodletting should not be practised after liquid effusion has taken place; it is contraindicated by the risk of unduly weakening the heart under these circumstances. It is not indicated when the symptoms denote only moderate acuteness or intensity of the inflammation.

In conclusion, as regards bloodletting, the mischief occasioned by its injudicious employment may greatly exceed the benefit ever to be expected from its judicious use. This only shows the great importance of discrimination; and the remark is alike applicable to this remedy in the treatment of other inflammations. But the injudicious abstraction of blood in pericarditis involves a peculiar source of danger. It has been seen that this disease destroys life by compression and paralysis of the heart. Any remedy which tends directly to weaken this organ, when weakness is the morbid condition to be most apprehended, can hardly fail, in proportion to the effect produced, to influence unfavorably the progress of the disease. This consideration cannot be too strongly impressed. Here, as in other affections, the attention of the practitioner must not be directed exclusively to the good which it is hoped may be effected by a potent remedy. The risk of harm is to be carefully weighed, and, with reference to the latter, the mode in which the disease tends to a fatal result is especially to be considered.

The foregoing remarks respecting bloodletting are essentially the same as in the first edition of this work. I have only to add, that during the ten years which have elapsed, I have had no experience as regards either the good or the evil results of bleeding in this disease. In none of the cases which I have observed during the period just named has this measure been employed.

Mercurialization.—Mercury given with the view of producing its special effects, or mercurialization, has been regarded, especially by British writers, as essential in the treatment of acute pericarditis. It has been supposed to exert a favorable influence on the progress of the disease by lessening the exudation of lymph and promoting the absorption of the inflammatory products. With reference more particularly to the first of these ends, it has been deemed important to induce mercurialization as early in the disease as possible. Calomel may be given for this purpose, either in fractional doses, repeated at short intervals, or in large doses, combined with sufficient opium to prevent its purgative action; and, in order to effect this object as speedily as possible, some writers have advised, in addition, inunction with mercurial ointment, or the mercurial vapor-bath. Bouillaud, and most French writers, on the other hand, have attached little importance, or none whatever, to the special effects of mercury in this disease. It has been claimed by some British writers that the disease is managed much more successfully in Great Britain than in France, and that this greater success is owing, in a great measure, to the free use of mercury. The testimony, however, of some British observers, on this score, is unfavorable to the efficacy of the remedy. Dr. Taylor, for example, found that in a considerable number of cases in which salivation was induced, a speedy abatement of the disease did not take place, and in several instances it was increased in extent and intensity.[1] With reference to this and other therapeutical questions, satisfactory statistical data are wanting. Nor is much to be expected from statistics in determining the value of remedial agencies in a disease which varies so greatly in severity and danger, according to its pathological relations and other circumstances, and which is not sufficiently common for a large number of cases to fall under the observation of any one practitioner. Moreover, here, as with regard to many other acute affections, the natural tendency of the disease, uninfluenced by remedial measures, is not fully ascertained.

Mercury is advocated in pericarditis on precisely the same grounds as in other inflammations—for example, pleurisy or pneumonia. But confidence in the utility of the remedy in the latter affections has of late years very greatly diminished, and at the present moment, most judicious practitioners do not deem its special effects in the treatment of these affections either demanded or desirable. Distrust of the supposed influence of mercurialization upon the process of exudation and the removal of morbid products, is evidently gaining ground. Even iritis, the affection which has been heretofore regarded as affording convincing ocular proof of the power of mercury in effecting the removal of lymph, has been shown by Dr. Williams, of Boston, to progress quite as favorably, if not more so, when this remedy is withheld.[1] Moreover, as regards the influence of mercurialization on pericarditis, the fact that, in the course of acute rheumatism, the disease has been repeatedly observed to become developed during salivation, militates against the applicability of mercury as a remedy. Fuller gives several instances in illustration of this fact, and examples have fallen under my own observation. Confessing doubt concerning the propriety of mercurializing patients affected with pericarditis, I am not prepared to deny, *in toto*, the importance of this method of treatment, inasmuch as the inconvenience and evils incident to moderate salivation, in many cases of this disease, are hardly deserving consideration, provided the remedy be entitled to a tithe of the value which has been heretofore claimed for it.

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[1] Bellingham, op. cit., pt. ii, p. 326.

Assuming a certain amount of efficacy in behalf of mercurialization, it is certainly not indicated in all cases. It is allowable chiefly in cases of idiopathic and rheumatic pericarditis. It is contraindicated by the coexistence of Bright's diseases, and is not appropriate whenever pericardial inflammation is developed in connection with anæmia or a broken constitution. The importance of this discrimination is conceded even by those who advocate strongly the importance of the remedy.

The foregoing remarks on mercurialization are retained mainly as they appeared in the first edition of this work. My experience, as regards the use of mercury in this disease, during the last ten years, is quite limited. I have notes of two cases which were under observation simultaneously, both being cases simply of rheumatic pericarditis, the severity in each being apparently the same. Of these two cases one was treated exclusively with opium, and in the other case mercury was given to the extent of producing ptyalism. In the case treated with opium, the patient rapidly improved, and was discharged after having been only thirteen days in hospital. In the case treated with mercury, the improvement was slow and the convalescence tedious. The latter case was not under my care. I noted it with reference to the contrast with the preceding case as regards the difference in progress. In another case which I saw in consultation in private practice, the patient was a lad, twelve years of age. The pericarditis occurred coincidently with rheumatism affecting a knee and a shoulder-joint. Small doses of mercury were given until the gums were slightly touched. Up to the time of the mercurialization the case had progressed satisfactorily; but, within a week afterward, pleurisy occurred successively in both sides of the chest, accompanied by abundant effusion, which, however, disappeared pretty rapidly, and the patient recovered. I introduce these cases as embracing my experience in the use of mercury in pericarditis since the publication of the first edition of this work.

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[1] On the Treatment of Iritis without Mercury. By Henry W. Williams, M.D. Reprinted from the Boston Medical and Surgical Journal, 1856. The conclusions in this paper are based on the results of the treatment of sixty-four cases of iritis without mercury. These results are of great interest and value in their bearing on the non-mercurial treatment of inflammations generally.

Sedatives.—Under this head, I refer to certain drugs which depress the powers of the system, and particularly the action of the heart, such as antimony and the veratrum viride, and others which diminish the frequency of the heart's action without necessarily depression; the latter are digitalis and aconite. The excitement and disorder of the circulation often incident to the first period of pericarditis might perhaps suggest the employment of depressing sedatives. In general, they are not appropriate. Employed during the period of effusion, especially if the quantity of liquid be large, they are dangerous remedies if carried to the extent of weakening the movements of the heart. The cardiac sedatives which diminish the frequency of, without weakening, the heart's action are not open to the objections which apply to the use of the sedatives which may be distinguished as depressing. I am not able to bear testimony to their value in the treatment of pericarditis, from lack of experience in observing their effects. In the early period of the disease, that is, before liquid effusion, aconite is rationally admissible; and it is reasonable to consider digitalis applicable to a later period, when the action of the heart may be feeble and irregular, on the ground of the utility of this remedy in cases of dilatation and fatty degeneration, the effect of the remedy being that of a cardiac tonic.

Revulsives or Counter-irritants.—These are useful for the same reasons, and probably to the same extent, as in the treatment of inflammation affecting analogous structures, for example, pleurisy; and the general principles which should govern their employment are the same. During the early and most acute period of the disease, blisters and other active modes of counter-irritation are inadmissible. Revulsive measures, such as sinapisms, fomentations, and foot-baths, are, to a certain extent, useful during this period. Vesication over the præcordia when the pericardial sac is distended, and the intensity of the inflammation has abated, probably hastens absorption, as it apparently does in cases of pleurisy with effusion. But there is this objection to the application of blisters directly over the heart: they interrupt daily physical exploration in order to determine the quantity of liquid effusion, &c. After the absorption of the liquid, it is possible that moderate counter-irritation may contribute in some measure to bring the inflammation speedily and completely to an end, and prevent a termination in the chronic form of the disease.

Opium.—The utility of opium is generally admitted. But there are grounds for the belief that the value of this remedy in the treatment of pericarditis has not been, and is not, generally appreciated. In analogous local inflammations—peritonitis, pleuritis, pneumonia, and perhaps may be added meningitis—opium exerts often a remarkable influence, as not merely a palliative, but a remedial agent. It relieves pain and quiets functional excitement; but, more than this, it appears to control, to a considerable extent, the processes of inflammation, abating its intensity, abridging its duration, and contributing to a favorable termination. To secure its full potency, it must hold a leading, not a subordinate, place in the management; and clinical experience shows that in these affections there is often a remarkable tolerance of the remedy, so that, to produce a proper effect, large doses may be requisite. Analogy would lead to the expectation of a similar remedial power in cases of acute pericarditis. It remains to accumulate a sufficient number of cases in the treatment of which reliance has been chiefly placed on the free use of opium, in order to confirm the correctness of this inferential reasoning. It will be understood that these remarks have reference to opium as a prominent remedy in the treatment of pericarditis. As a subsidiary remedy, its value is already sufficiently attested by experience.

The use of opium has this advantage over other measures, namely, it is not positively contraindicated by any of the various and opposite circumstances associated with the disease. If it be appropriate in rheumatic pericarditis, it is appropriate also when pericardial inflammation is developed in the course of renal disease, or in other pathological relations. If it be not efficacious, it is not mischievous, except so far as it may supplant other measures and involve loss of time. It admits of being employed tentatively without incurring much, if any, risk of doing harm, even by delay. Its potency for evil is not proportionate to its potency for good, and this cannot be said of most potent remedial agencies.

The periods of the disease most favorable for the beneficial influence of opium are the period anterior, and that subsequent to, liquid effusion. It is, however, by no means certain that the remedy affects unfavorably absorption of the effused liquid. I have known an abundant pleural effusion, in connection with pleuro-pneumonia, to disappear rapidly when no other remedy was employed.

My experience for the last ten years has tended to confirm the correctness of the foregoing remarks on the use of opium in this disease. In several of my cases this was the only remedy employed.

Stimulants.—In all inflammatory affections, diffusible or alcoholic stimulants form an essential part of the treatment whenever measures to sustain the power of the system and obviate the tendency to death by asthenia are indicated. Pericarditis is by no means exempt from these indications; on the contrary, since the immediate danger from the disease chiefly arises from weakness of the heart, sustaining measures would seem to be called for earlier and more imperatively than in most local inflammations. As remarked by Stokes, little is said by authors on the use of stimulants in pericarditis. This distinguished author adds: "I am convinced that cases are often lost from want of simulation at the proper time; and it is certain that, in every case of dangerous pericarditis, after the first violence of the disease has been subdued, we should be anxiously on the watch for the moment when the weakened heart requires to be supported and invigorated." In the treatment of inflammatory affections generally, timidity in the use of stimulants is apt to proceed from the attention of the practitioner being too exclusively directed to the local morbid processes, the state of the system being overlooked, or not sufficiently regarded. Measures designed to abate the intensity of inflammation, and to control its processes, pertain, for the most part, to an early period in the disease. After a certain time, all the immediate local results of inflammation, which may be expected to occur, have already taken place, and the general object of treatment, then, is to maintain the forces of life through the processes of restoration. The physician, under these circumstances, is to regard the patient more than the disease, in looking for therapeutical indications. Alcoholic stimulants, given to support the flagging powers of life, do not excite, as in health, but, associated with nutriment, they sustain the vital forces, keeping the patient alive, in some cases in which death from asthenia is threatened, until the period of danger is passed. They are to be given with a freedom proportionate to the indications and the apparent effects. These general views, as applied to local inflammations indiscriminately, seem to me to possess very great practical importance. With regard to pericarditis, it is only necessary to add that they are at least as applicable to this as to any other inflammatory affection.

Some of the cases which I have observed since the foregoing remarks were written, appeared to exemplify in a striking manner the efficiency of stimulants given freely with reference to the objects just stated. Among my cases ending in recovery are two in which the pericarditis was rheumatic, and associated also with pneumonia. In one of these cases the pneumonia was double. It seemed fair to attribute the recovery in these cases, in a great measure, to the free use of alcoholic stimulants.

Eliminatives.—The propriety of eliminative remedies in pericarditis rests on the pathological relations of the disease in certain cases. As developed in connection with rheumatism and renal disease, the pathology is supposed to involve a *materies morbi*, the removal of which from the system may be promoted by remedies employed for that purpose. Thus, in rheumatic pericarditis, the question arises whether remedies supposed to be efficacious as eliminative agents in rheumatism, may not diminish or expel a cause which serves to keep up the cardiac inflammation. It is obvious that remedies of this class, if they possess any efficacy, are more indicated as prophylactic than curative agents; and, with reference to the prevention of pericarditis in rheumatism, clinical observation shows that cardiac inflammation may become developed under any of the various methods of treatment which are pursued in the latter affection. At the present time, the introduction of alkalies (the bicarbonate of potassa or soda), largely and promptly, is much relied upon for preventing both pericarditis and endocarditis. It is claimed by Fuller that these cardiac affections may be prevented with certainty if the alkaline treatment, in cases of rheumatism, be quickly carried to the extent of producing alkalinity of the urine. Clinical observation demonstrates that this treatment has a positive prophylactic influence as regards these affections. In proof of the correctness of this statement, the statistical researches of Dr. W. H. Dickinson may be cited: Of 48 cases in which "full alkaline treatment" was employed, that is, in which the quantity of the alkaline salts given exceeded four drachms daily, the heart was affected in but one case. Of these 48 cases, the full alkaline treatment was employed alone in 22, and conjoined with other measures in 26. In not one of the latter was the heart affected. On the other hand, of 110 cases treated either with other measures, namely, venesection, nitre, mercury, iodide of potassium, &c., or with a partial alkaline treatment, the heart was affected in 35.[1] Cardiac inflammation may become developed even after the alkaline treatment has been fully employed. Within three months from the time I am writing, I have seen three cases in private practice in which pericarditis occurred after alkalinity of the urine had been produced. But admitting a positive and a considerable prophylactic influence of alkalies, given largely and promptly, it is reasonable to suppose that, within certain limits, after the development of pericarditis, they are useful by preventing the continued operation of the causative agent in the blood, and which produced the inflammation. It may be that the alkalies in rheumatism exert their effect by neutralizing the *materies morbi*, rather than by elimination, but it is probable that they form combinations which are readily thrown off from the system. As regards the extent to which alkalies should be given after cardiac inflammation (pericarditis or endocarditis) has been developed in the course of rheumatism, it is not desirable nor proper to continue the full alkaline treatment. It suffices to continue the use of alkalies in such doses as may be required to keep up the alkalinity of the urine.

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[1] *Vide* art. by Dr. Dickinson in Med.-Chir. Trans., of Royal Med and Chirurg. Society, vol. xlv, 1862. Also, Braithwaite's Retrospect, July, 1869, and the Half-Yearly Abstract by Ranking, July, 1869.

When pericarditis is developed in connection with Bright's diseases, the morbid material is supposed to be urea, or the products of its decomposition in the blood. The remedies supposed to act as eliminatives are diuretics, cathartics, and sudorifics. As regards the employment of these remedies with a view to elimination, in renal pericarditis, it is important to remark that they are contraindicated, not only when they come into opposition to other measures of greater importance, but when, irrespective of their eliminative operation, they are likely to prove hurtful. This remark applies more particularly to active purgative remedies. The probability of good being effected by means of elimination is not sufficient to warrant taking the risk of doing injury. Remedies to act on the kidneys and skin may be given more safely. As a sudorific, the hot air or vapor bath is preferable to other means. In addition to their greater efficiency, they have these important advantages over the water bath, namely, they may be taken with the head moderately low, and without exertion on the part of the patient.

Having noticed the more important of the therapeutical measures embraced in the treatment of acute pericarditis, the practical points which have been presented may be recapitulated, and others added, in considering briefly the indications which belong, respectively, to the successive periods of the disease, viz., prior to, during, and after liquid effusion.

Treatment prior to liquid effusion.

The chief objects of treatment in this period are, abatement of the intensity of the inflammation, limitation of the products of inflammation (serum and lymph), and, perhaps, by effecting these objects, shortening the duration of the disease. The means which may be employed for these ends are, bloodletting, mercurialization, opium, and eliminatives.

Bloodletting is admissible only in cases of idiopathic, and in certain cases of rheumatic pericarditis. It is contraindicated by coexisting renal disease, anæmia, feebleness, or a broken constitution. It is never indicated when pericarditis occurs in connection with the eruptive or continued fevers, pyæmia, &c. It should be employed only when the inflammation has a certain degree of intensity, as shown by febrile movement and a firm pulse. The repetitions of bloodletting, and the amount of blood withdrawn, are to be determined by the symptoms and the effect, bearing in mind the danger of weakening too much the heart by this measure. It is never to be employed when physical exploration of the chest affords evidence that effusion of liquid has taken place.

The propriety of mercurialization in any case is questionable. If ever appropriate, it is admissible only in cases of either idiopathic or rheumatic pericarditis, and when the constitution of the patient is not greatly impaired from any cause. Anæmia constitutes a contraindication. If this measure be employed, the system should be brought rapidly under the effects of mercury, but it is never necessary to induce severe ptyalism; on the contrary, this is to be avoided, if possible. Patients with this disease are often mercurialized with difficulty. When this is found to be the case, it is certainly better to relinquish the attempt, than to introduce a quantity of the remedy into the system, which may, in the end, lead to excessive effects.

Opium is safer and more reliable than either bloodletting or mercurialization. It may be employed in the cases in which these measures are contraindicated; and it may be employed in conjunction with them. It should be given in doses sufficient to relieve pain and tranquillize the circulation. The doses required to produce a sufficient effect will sometimes be large, owing to the tolerance of the remedy in this disease.

Eliminatives are indicated in cases in which the disease is dependent on rheumatism or uræmia.

Treatment during liquid effusion.

The chief objects of treatment in this period are, the prevention of further accumulation of liquid; the promotion of its absorption; the invigoration of the heart and forestalling danger from paralysis and compression of the organ. The means for these ends are, opium, counter-irritation, stimulants, nutritious diet, and to these may be added, diuretics and hydragogue cathartics.

Indications for opium may be present in the second, as well as in the first period, but not to the same extent. To relieve pain and quiet irritation are still objects of treatment, and this remedy is indicated in doses sufficient to effect these objects.

Vesication upon or near the præcordia, which is not admissible in the first period, is now useful in promoting absorption. In order not to interfere with physical examinations of the chest, by means of which the progress of the disease, from day to day, is ascertained, it is preferable to apply blisters in the neighborhood of the præcordia, and not directly over the heart. The employment of a series of blisters applied in different situations, allowing the blistered surface to heal as quickly as possible, is best suited to promote absorption.

Diffusible stimulants are indicated, in the second period, by weakness of the heart. They are indicated in proportion as the heart becomes weakened by paralysis and compression. Weakness of the heart is manifested by the pulse and other symptoms; but more distinctly by physical signs. Feebleness or suppression of the apex-beat, diminished intensity of the heart-sounds, more especially of the first sound, and extinction of the latter, call imperatively for measures to invigorate the heart. Alcoholic stimulants, in the form of either wine or spirits, are the most efficient means for this end. They should be given as freely as they are found to be well borne, the criterion being, not a certain quantity, but a certain effect. The desired effect is increased strength, with diminished frequency, of the pulse. The physical signs also afford a guide in regulating the quantity of stimulants. The reappearance or increased force of the apex-beat, and an approximative return of the sounds of the heart to their normal relative intensity—in other words, improvement in the first sound more particularly—denote the beneficial influence of stimulation.

In conjunction with stimulants, a nutritious diet is indicated. Whenever stimulants are useful, the diet cannot be too nutritious. Animal essences and tender meat constitute the diet which is most sustaining. But those articles of food are of course to be selected which are best adapted to the digestive powers in individual cases, and this is to be determined by experimental trials.

Digitalis may be employed in this stage when the action of the heart is notably feeble, frequent, or irregular.

Diuretics and hydragogue cathartics may sometimes be employed with advantage, with a view to promote absorption of the effused liquid. But the employment of these remedies demands great circumspection. They are contraindicated whenever stimulants and sustaining diet are required. This caution has reference more to hydragogue cathartics than to diuretics, but measurably to the latter.

Eliminative remedies may be continued, under proper restrictions, into the second period. They must not conflict with other measures upon which greater reliance is to be placed.

Aside from the indications just mentioned, purgative remedies are not advisable in the treatment of pericarditis, save to obviate discomfort attending constipation.

It will not be amiss to repeat the caution not to permit much exertion on the part of the patient, when the heart is compressed by an abundant accumulation of liquid effusion. Sudden and fatal syncope may be induced by the effort of rising from the bed to go to stool, as in an instance, referred to more than once, which came under my observation.

The importance of daily or frequent explorations of the chest, in order to determine the progressive diminution of the liquid effusion, and its final disappearance, has been repeatedly referred to, but it cannot be too strongly enforced. It is also to be borne in mind, that even where the symptoms denote acuteness of the pericardial inflammation, there is not always much liquid effusion. The indications in the second stage, therefore, which relate to the presence of liquid, will hinge on the information to be obtained by means of physical signs.

Treatment after the absorption of liquid effusion.

The chief object of treatment in this period is to promote the complete disappearance of inflammation. The means for this end are, counter-irritation, tonic remedies, an invigorating diet and regimen.

It is perhaps probable that moderate counter-irritation near the præcordia tends to expedite the final cessation of inflammation, and to prevent it from becoming chronic. The modes of counter-irritation suited to this object are blisters, issues, and pustulation with croton oil. The counter-irritation should be so restricted in degree as not to produce constitutional disturbance, nor to prove a source of exhaustion. Without this precaution, the evils would be likely to overbalance the good effect.

In acute inflammations generally, the local processes of restoration go on more rapidly, the liability to relapse is less, and the final recovery is more complete, in proportion as the general powers of the system are invigorated. Hence, one great advantage in not employing unnecessarily, during the progress of the disease, debilitating measures of treatment. The less the patient is enfeebled, the more speedy and safe the convalescence. Hence, tonic remedies, a nutritious diet, cheerful relaxation of mind, and gentle exercise in the open air, are important as soon as convalescence is established. These are the means by which the body regains strength and vigor. They are applicable to pericarditis as well as to other inflammations. While undue exertion of body or mind, imprudent exposure, and excesses of all kinds, are, of course, to be avoided, the regimen and diet best calculated to restore or improve the general health will affect most favorably the condition of the organ recently inflamed.

An effect of undue physical exertion, in this disease, before the pericardial surfaces have become firmly adherent, is the breaking up of the adhesions to a greater or less extent. This is followed by pain and soreness, and the convalescence is retarded. In a case under my observation, on two occasions, directly following an unusual muscular effort, in conjunction with a renewal of pain and soreness, the pericardial friction-murmur, which had ceased, returned and peristed for several days.

In the treatment of pericarditis, associated affections, especially endocarditis, pleurisy, and pneumonia, will often claim attention. The measures to be directed to these affections need not be here considered. The treatment of the pericarditis, it is obvious, must be more or less modified under these circumstances. As a rule, they enforce greater circumspection in the use of debilitating remedies, and call for an earlier and more efficient employment of sustaining measures.

The danger in cases of pericarditis is much enhanced when the disease is complicated with notable disorder of the nervous system, giving rise to active delirium, convulsions, &c., symptoms which, it has been seen, often mask the cardiac affection. It is not easy to decide, with our present knowledge, as to the measures most likely to prove successful in this class of cases. In the case under my observation which ended in recovery, the treatment consisted of the free use of alcoholic stimulants, sustaining diet, and a blister upon the præcordia.

SUBACUTE AND CHRONIC PERICARDITIS.

Pericarditis may be subacute without becoming chronic. The inflammation may be so slight as to give rise to few, if any, local subjective symptoms, and to very little constitutional disturbance. The following cases will serve to illustrate the latency of this variety of the disease:

Case 1. A seamstress, aged 24, was attacked with subacute rheumatism affecting the ankles, knees, wrists, and elbows. The rheumatic affection was not sufficient to cause her to take to the bed. On the third day after the attack she began to take the bicarbonate of potassa in doses of a drachm every two hours. On the day following, there was a well-marked pericardial friction-murmur. This murmur continued for four days. There were no signs of pericardial effusion. She had slight pain referable to the heart. She took to the bed reluctantly. The bicarbonate of potassa was continued for several days, with an anodyne at night, and afterward quinia was prescribed. The rheumatic affection lasted for ten days.

Case 2. A girl, aged 8 years, brought to my clinic at the Long Island College Hospital, by my colleague, Prof. Gilfillan, a fortnight previously had been seized with pain in both knees. The pain disappeared on the next day, and she complained of pain in the præcordia. Prof. Gilfillan had seen her three days before bringing her to the clinic. She had not been confined to the bed. Prof. Gilfillan had found well-marked pericardial friction-murmur, together with a mitral systolic murmur. These signs were still present, and the case served to illustrate them to the medical class. There was some pain and tenderness in the præcordia, and the respirations were somewhat accelerated. The patient, however, was up and about, scarcely appearing to be ill. There were no signs of pericardial effusion. The patient recovered without any untoward symptoms.

In cases like these auscultation reveals unexpectedly the existence of pericarditis; it would hardly be suspected from the symptoms. There is, however, always a liability to liquid effusion, in more or less abundance, no matter how slight may be the inflammation. There is no more a fixed relation between the intensity of the disease and the quantity of liquid effused in pericarditis than in cases of pleurisy; the effusion may be slight when the inflammation is intense, and it may be copious although the pericarditis be notably subacute. Such cases, therefore, claim watchfulness and care chiefly with reference to the liability to the effusion of liquid. It is to be added, that the friction-murmur in these cases may be loud. The intensity of this murmur is by no means a criterion of the intensity of the disease. Indeed, since in cases of subacute pericarditis the power of the heart's action may be but little diminished, the friction-murmur, other things being equal, will be louder than when the muscular power of the heart is weakened by the paralyzing effect of the inflammation.

In chronic pericarditis the disease may have been from the commencement subacute. In these cases, when the attention is first directed to the chest, the pericardial sac is sometimes found already largely distended. If death occur during the period of effusion, the liquid is found to be moderately or slightly turbid, a small quantity of lymph adhering, in circumscribed patches, to the pericardial surfaces. The disease is analogous to chronic pleurisy, as the latter is not infrequently presented in medical practice. In other cases, this variety of the disease succeeds the acute form. In acute pericarditis, if the inflammation do not disappear in the course of from two to three weeks, the disease may be considered as having become chronic.

The anatomical conditions in chronic pericarditis, more especially when it follows an acute affection, are quite different in different cases. It suffices to arrange these differences into two classes, viz., *First.* Absence of liquid effusion, and the pericardial surfaces agglutinated by interposed layers of lymph, of variable thickness, without, or with only imperfectly organized attachment; *Second.* More or less distension of the pericardial sac with liquid, which is turbid, puruloid, or even truly purulent.

Upon these two classes of anatomical conditions may be based a division into chronic pericarditis with, and chronic pericarditis without, the accumulation of liquid.

In chronic pericarditis without the presence of liquid, adhesion of the pericardial surfaces by organized attachment is prevented by the abundance of lymph. The latter often forms a series of layers, which may be successively peeled off from the surface of the heart. These layers are dense, resembling membranous structure, but they are not organized. The pericardial surface beneath presents the appearances of inflammation, and frequently in detaching the layers of lymph, small circumscribed collections of sero-purulent liquid are discovered. The adherence of the layers of lymph to the visceral and parietal surfaces of the pericardium, and to each other, may be quite firm, but it is only by mechanical agglutination. The deeper layers of lymph are often colored with hæmatin.

A low grade of inflammation is probably kept up by the presence of the lymph in these cases, which acts like a foreign substance, or, successive attacks of subacute inflammation are frequently renewed. These cases are hopeless as regards ultimate, complete recovery, applying the term recovery to the cessation of inflammation, with the occurrence of organized adhesion of the pericardial surfaces, to a greater or less extent.

In chronic pericarditis with effusion, the accumulation of liquid is often much greater than in cases of the acute form of the disease. The pericardial sac, from long-continued distension, yields to the pressure, becomes more or less dilated, and the amount of effusion in some instances is enormous. Perhaps the most remarkable case of large accumulation on record was observed by Prof. Alonzo Clark, and reported by the late Dr. Swett.[1] Death occurred in ten weeks from the date of the attack, and on examination post mortem "the pericardium was found to occupy the whole anterior part of the chest, pushing the diaphragm downwards so as to form a very large convexity towards the cavity of the abdomen. The liver was pushed downwards so that its upper convex margin reached the margin of the ribs, and both lungs were pushed into the posterior and lateral portions of the thorax. Had the entire contents of the pericardial sac been fluid, it could not have been less than ten pints; but, as it was, there was at least a gallon of clear, yellow serum." Cases in which the accumulation amounts to from two to three pints are not very uncommon. The sac, when dilated much beyond its normal capacity, becomes enlarged disproportionately in width. Its pyriform shape is not preserved, as it is in acute pericarditis. In proportion to its abnormal size, it occupies, of course, space at the expense of the pulmonary organs, and interferes with the thoracic and diaphragmatic movements in respiration.

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[1] Lectures on Diseases of the Chest, 1852, p. 394.

Chronic pericarditis without liquid effusion is often unattended by symptoms which point to the seat of disease. Acute pain is rarely present. A sense of uneasiness, constriction, or indefinite distress, may be referred to the præcordia; palpitation may be complained of with dyspnœa, on exertion; but there may be entire absence of all subjective symptoms referable to the heart.

The presence of an abundant liquid effusion will be likely to give rise to symptoms directing attention to the chest, but not distinctive of the disease. The symptoms are essentially those incident to the period of effusion in acute pericarditis, viz., palpitation, feebleness and irregularity of the pulse, præcordial distress, dyspnœa, and tendency to syncope, especially on exertion, lividity, œdema, &c. These symptoms may be less marked than in acute pericarditis, although a much larger quantity of liquid may be contained in the pericardial sac, the accumulation taking place more slowly and greater tolerance being acquired. But even when considerable effusion exists, the disease may be latent as regards subjective symptoms distinctly referable to the heart.

The physical signs, when liquid is not present, are not distinctive of existing chronic inflammation. Certain signs may be present denoting union of the pericardial surfaces, but not indicating the mode of this union. These signs will be presently noticed under the head of pericardial adhesions. Creaking friction-sound is occasionally discovered. The disease, in fact, under these circumstances, frequently does not offer strongly-marked diagnostic phenomena; and, without an acquaintance with the previous history, it is by no means easy to arrive at a positive diagnosis. Knowledge of the fact that acute pericarditis has recently existed, taken in connection with the symptoms and signs, is important with reference to the diagnosis. In these cases, when not preceded by acute pericarditis, or when they come under observation after the inflammation has ceased to be acute, the disease may be overlooked. If, as is often the case, valvular lesions and enlargement of the heart coexist, the symptoms and signs may be referred exclusively to these, pericarditis not being suspected.

An abundant liquid effusion gives rise to physical signs which have been already considered in connection with acute pericarditis. The signs do not differ from those in the latter affection, except so far as they are modified by the presence, often, of a much larger accumulation of liquid than occurs in the acute form of the disease. The space occupied by a largely dilated pericardial sac is, of course, proportionately greater than when the sac is merely distended; and the form of the sac being altered by its greater relative width, the area of dulness on percussion, corresponding to the space which it occupies, does not present the pyriform shape characteristic of the outline of dulness in acute pericarditis. The dulness on percussion, in proportion as the amount of liquid is greater, is more marked in degree, and if the amount be large there is absolute flatness. The auscultatory signs show removal of the lung on each side to a greater distance from the median line, the extent of the separation being in proportion to the dilatation of the sac. A friction-sound, in some cases, is discovered, notwithstanding a very large accumulation of liquid. The apex-beat is suppressed; but a diffused shock may be felt over the præcordia. The latter was observed in the case of enormous accumulation reported by Dr. Swett. It was observed in that case that the limits of dulness on percussion moved nearly an inch to the right or left, according as the position of the patient was on the right or left side, the extent of dulness undergoing no change. This variation in situation of the area of dulness, with change of position, in cases of pericardial effusion, has been observed in other cases. Enlargement or bulging of the præcordial region is more apt to be marked in chronic than acute pericarditis, in consequence of the larger collection of liquid. The depression of the diaphragm may be sufficient to cause marked swelling at the epigastrium, and even an unusual prominence of the abdomen. Undulation in the intercostal spaces is oftener observed in chronic than acute pericarditis. In the case reported by Swett, it was perceived in the epigastrium. The left lung is sometimes pressed upward, to a considerable height, above the clavicle. Stokes cites a case which came under his observation, in which a tumor was produced above the clavicle sufficiently large to produce great deformity of the neck. This tumor was present for several days; it was increased by coughing, and gave the pulmonary sound on percussion, with vesicular murmur and wheezing râle on auscultation. A similar case was observed by Graves. The enlargement of the chest and depression of the diaphragm, in cases of very large effusion, will occasion an obvious restraint of the costal and abdominal movements of respiration.

The differential diagnosis of pericardial effusion, based on the physical signs, involves the same points in chronic, as in acute, pericarditis, and these need not be repeated.

The objects of treatment in chronic pericarditis are the absorption of the morbid products, and the removal of the inflammation. Therapeutical measures having reference to these objects, are, vesication, and the use of certain remedies which are supposed to act as sorbefacients, of which the most prominent is iodine. Iodine has been supposed to act efficiently, in some instances, when applied externally. This method is recommended by Stokes.

But the treatment must be governed, in a great measure, by circumstances which have reference indirectly to the objects just stated, viz., the morbid conditions as respects the presence or absence of liquid; the vital condition of the heart, or, in other words, the weakness of the organ; coexisting affections, and the constitution of the patient. The solid products of inflammation, consisting of thick layers of condensed lymph, cannot be removed by sorbefacient remedies. More is to be expected from efforts to promote the absorption of liquid; but if the pericardial sac be much dilated, the prospect of success is small. In proportion to the weakness of the heart, stimulants are called for. Coexisting affections will claim appropriate attention, and the general condition of the system will be likely, on the one hand, to contraindicate therapeutical measures which tend to impair the vital forces, and, on the other hand, to indicate a sustaining course of treatment.

In cases of large dilatation of the pericardial sac with liquid which does not diminish under appropriate remedies and gives rise to distress and danger from compression of the heart and outward pressure on the adjacent parts, the propriety of puncturing the pericardium is to be considered. This operation has been repeatedly performed, with immediate relief of distressing symptoms, apparent prolongation of life, and in some instances it has been followed by recovery.[1] Assuming the diagnosis to be clear, and that other measures have proved ineffectual, paracentesis is certainly warranted, even as a means of temporary relief. Comparative comfort and postponement of a fatal result may reasonably be expected from the operation. That recovery is not to be looked for in the great majority of cases arises from the almost hopelessness of chronic pericarditis, irrespective of the danger incident to the quantity of liquid accumulation. Further observations may show that the operation is to be resorted to in cases in which the sac is largely distended or dilated, when the distress is not extreme and the danger not imminent. It may perhaps be shown by experience to be applicable to the treatment of acute as well as chronic pericarditis. The success with which paracentesis has been employed in cases of pleurisy, warrants a conjecture that the same measure may be extended equally to pericarditis with effusion.

In performing paracentesis of the pericardium, the method practised by Dr. Bowditch in cases of pleurisy is to be preferred. This method consists in the introduction of a small exploring trocar, to the canula of which is attached a suction-pump. The wound made by this instrument is trivial, the liquid may be withdrawn slowly, the quantity regulated by the immediate effects, and the operation repeated as often as may be deemed advisable. The trocar is to be introduced in the fourth or fifth intercostal space between the nipple and the sternum, the patient lying upon the back; the physical signs showing the pericardium to be in contact with the thoracic wall at the point of puncture, and the heart removed from the walls at that situation. Aran, of Paris, reported a case in which a solution of iodine was injected, after the removal of the liquid, with apparent benefit.

PNEUMO-PERICARDIUM AND PNEUMO-PERICARDITIS.

Air or gas gains access within the pericardium by means of fistulous communications with the stomach, œsophagus, and the pulmonary organs; or through wounds of the chest perforating the pericardial sac; and in rare instances it may possibly be due to decomposition of liquid products within the sac. Inflammation, with more or less liquid effusion, is almost invariably present. The affection is then properly designated pneumo-pericarditis. It is analogous to that variety of pleurisy which is commonly known as pneumo-hydrothorax. But inflammation is not necessarily present. In a case related to me by Dr. Knapp, of Louisville, to which reference has before been made, a patient was stabbed with a knife, which penetrated the pleural cavity and perforated slightly the pericardium. A splashing sound with the heart's action was immediately heard, which continued for a few days and disappeared. The symptoms and signs, subsequently, did not denote pericarditis, but the patient had pleurisy, which was followed by considerable contraction of the left side. The splashing sound, in this case, was fairly attributable to the presence of air and probably a little blood within the pericardium. The recovery was complete, and the patient was examined by me some two or three years after the injury. In such a case, the affection, assuming that inflammation was not present, is properly called pneumo-pericardium.

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[1] For a *résumé* of cases which have been reported, numbering thirteen, the reader is referred to Bellingham's work, pt. ii, p. 330. For report of a case in which this operation was successfully performed, see Boston Med. & Surg. Journ., Feb. 28, 1867. For report of case of hemorrhagic pericarditis, in which paracentesis was twice employed with marked relief by Henri Roger, see Half-Yearly Abstract of Medical Sciences, Amer. ed., July, 1869.

Stokes relates a case in which the coexistence of liquid and gas was predicated on the peculiar auscultatory phenomena. The account is best given in his own words. "The patient was a young man of lymphatic temperament, who had labored under an attack of acute pericarditis for a few days before I saw him. On my first examination he presented the usual signs of dry pericarditis, with a considerable effusion of lymph of the ordinary consistence. The rubbing sounds, though loud and distinct, had nothing unusual in their character, and the patient suffered but little distress. After two or three days I saw him again, and found that his state had become very much altered. His appearance was haggard and worn, and he complained of extreme exhaustion, which he attributed to a total deprivation of sleep. This was induced by the extraordinary loudness and singular character of the sounds proceeding from the cardiac region; for though up to this period the rubbing sounds were distinctly perceptible by means of the stethoscope, the patient was quite unconscious of their existence. They had suddenly, however, become so loud and singular, that the patient and his wife, who occupied the same apartment, were unable to obtain a moment's repose. On examination, a series of sounds was observable which I had never before met with. It is difficult or impossible to convey in words any idea of the extraordinary phenomena then presented. They were not the rasping sounds of indurated lymph, nor the leather creak of Collin, nor those proceeding from pericarditic with valvular murmur, but a mixture of the various attrition murmurs with a large crepitating and a gurgling sound, while to all these phenomena was added a distinct metallic character. In the whole of my experience I never met with so extraordinary a combination of sounds. The stomach was not distended with air, and the lung and pleura were unaffected, but the region of the heart gave a tympanitic *bruit de pot fêlé* on percussion; and I could form no conclusion but that the pericardium contained air in addition to an effusion of serum and coagulable lymph. In the course of about three days the signs of air disappeared, leaving the phenomena as they were at the first period of the case. The convalescence of this patient was slow, and the rubbing sounds continued for an unusual length of time. His recovery was ultimately perfect."[1]

Stokes, in connection with this case, cites two additional cases, one reported by Graves, and the other communicated by Dr. B. McDowel. In Graves's case, pericarditis was induced by the opening of an hepatic abscess into the pericardial sac. The case proved fatal, and after death it was ascertained that the abscess also communicated, through a fistulous opening, with the stomach. The gas contained within the pericardium was derived from the stomach passing through the hepatic abscess. The patient presented over the præcordia friction-sounds, with an occasional metallic click, giving the idea of a fluid dropping in the pericardium. Afterwards the sounds assumed the character of an emphysematous crackling. In McDowel's case, a fistula was found, after death, to have become established between the pericardial sac and a small anfractuous cavity in the right lung. A current of air through the trachea was observed to rise through the fluid contained in the pericardial sac, and the latter, when opened, contained air. Over the left side of the chest, in this case, auscultation discovered metallic tinkling, and splashing of fluid caused by the action of the heart.

In a case reported by Walshe, in which a communication existed between the œsophagus and pericardium, produced by the effort to swallow a knife, tympanitic resonance on percussion over the præcordia was marked, but neither a splashing noise nor metallic tinkling were observed. A distinctive phenomenon in this case "consisted in the change of position of tympanitic and dull percussion-sound, within the area of the cardiac region, according as the posture of the patient was changed from one to the other side."

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[1] On Diseases of the Heart and Aorta, Amer. ed., p. 38.

These cases are of much interest as showing the physical signs distinctive of the presence of air or gas and liquid within the pericardium. The auscultatory signs which may be expected to be present are, metallic tinkling sounds, and a splashing or gurgling noise, produced by the action of the heart. Their connection with the heart is to be determined, if there be room for doubt, by requesting a momentary holding of the breath. They are not, however, invariably present, as shown by the case reported by Walshe. Tympanitic resonance on percussion over more or less of the præcordia is marked. In the case observed by Stokes, a distinct *bruit de pot fêlé* was observed; and in Walshe's case variation in the relative position of tympanitic resonance and dulness, with change of posture. The production of a peculiar noise, so loud as not only to be heard by the patient and others, but to prevent persons from sleeping in the same apartment, is a remarkable and highly distinctive feature in Stokes's case.

The physical signs, in connection with the history and symptoms, seem to be amply sufficient for the diagnosis. There is a possibility that considerable distension of the stomach with gas and liquid may give rise to acoustic phenomena resembling those produced in some cases of pneumo-pericardium. But the evidences of pericarditis with effusion, under these circumstances, will be wanting. Cardiac gastric sounds, probably, require for their production that the pericardial sac shall be free from liquid. Again, metallic tinkling, and, possibly, splashing sounds may be produced by the action of the heart in some cases of pneumo-hydrothorax; but it is sufficiently easy to exclude the latter affection by the absence of its diagnostic signs.

The treatment of this variety of pericarditis does not claim distinct consideration.

PERICARDIAL ADHESIONS.

Inflammation of the pericardium, ending in recovery, involves, as has been seen, the formation of new tissue which serves as a medium of permanent union of the opposed pericardial surfaces. Pericarditis, when general, that is, when the inflammation extends over the whole, or the greater part of the membrane, is followed by this result, as inflammation of the pleura is followed by pleuritic adhesions. The pericardial adhesions now referred to, differ from those which have been considered as incident to a variety of chronic pericarditis. The latter are due to strata of lymph interposed between the surfaces of the pericardium, to which each pericardial surface becomes agglutinated. The lymph is unorganized, and is, in fact, equivalent to a foreign substance, at once separating and binding together, mechanically, the parietal and visceral portions of the pericardium. Under these circumstances, the pericardium is rarely, if ever, in a healthy condition. In adhesions by means of new tissue, the mode of union is quite different. It is by an organized attachment. The new structure, when formed, becomes thereafter an integral portion of the organism. These adhesions are not incompatible with a healthy state of the pericardial membrane; they do not necessarily constitute a disease, although they are the effects of disease. They become more and more firm with age. Some idea may be formed of the length of time after their formation, by the force required for their separation. It is customary to speak of them as more or less ancient. It is doubtful whether recovery from general pericarditis ever takes place without leaving more or less of these effects. The adhesions may be general or partial; in other words, the surfaces may be united over the whole heart, or only over a portion of the organ. General adhesions appear to be of much more frequent occurrence than partial. Of 70 cases analyzed by Louis, the adhesions were general in 60, and partial in 10; and of 86 cases analyzed by Dr. Chambers, 51 were universal, 4 nearly so, and 29 partial.[1] When the adhesions are general and close, the pericardium seems to be wanting, and it is conjectured that some of the cases reported by the early anatomists of absence of the pericardium, may have been cases of this description.

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[1] Decennium Pathologicum, Brit. and For. Med.-Chir. Rev., vol. xii, 1853. Also Bellingham, op. cit.

Pericardial adhesions are found, on examination after death, associated, in a certain proportion of cases, with valvular and other cardiac lesions. They are also found, not infrequently, when there had been no suspicion of cardiac disease. They denote, of course, that pericarditis has existed at some period during life, and this period may have been more or less remote from the time of death. They constitute, in a certain proportion of cases, the only abnormal condition which the heart presents. The practical questions connected with the subject are: What effects do they produce upon the heart and circulation, and how is their existence to be ascertained during life? These questions, it is obvious, are of considerable importance practically. They suggest the most convenient arrangement for the consideration of the subject.

What effects are produced by pericardial adhesions upon the heart and circulation?

Pathologists, for the most part, up to the present time, have regarded adhesions of the pericardium as constituting a very serious abnormal condition. Laennec held a contrary opinion, considering them as often harmless. Among recent writers on diseases of the heart, Bouillaud concurs in the opinion of Laennec; but the doctrine inculcated by Hope, was that they inevitably lead to enlargement of the heart, and, sooner or later, to a fatal result. Investigations since the publication of the last edition of the treatise by Hope show, conclusively, that this distinguished author was led to exaggerate the evils and dangers attendant on the remote consequences of pericarditis.

Pericardial adhesions, general, and evidently of long standing, are found, not infrequently, when, in all other respects, the heart presents a normal, healthy appearance. Cases exemplifying the correctness of this statement have been reported by Bouillaud, Stokes, King, Barlow, W. T. Gairdner, and others.[1] The following instance, which I find among my recorded cases, will serve as an illustration: A male patient, aged 35, was admitted into the hospital in a state of unconsciousness, and died fifteen hours after admission. The previous history was not ascertained. On examination after death, morbid appearances were found denoting meningitis. The heart presented universal, old adhesions. Considerable force was required in separating the pericardial surfaces. The organ was apparently not enlarged. It weighed 10½ ounces. The walls and cavities were normal, and the muscular substance was not altered in color or consistence. In this instance, that the adhesions were of ancient date is inferred from their firmness. Stokes refers to a case which came under his observation, in which, death occurring seven years after an attack of pericarditis, the patient for several years exhibiting no symptoms of heart disease, the pericardial sac was found to be obliterated, and the heart otherwise in a perfectly natural condition. The occurrence, in even a small number of cases, of adhesions which, after several years of exemption from all cardiac trouble, are found not to be accompanied by any other abnormal condition of the heart, suffices to show that they do not necessarily give rise, as was stated by Hope, to serious effects upon the heart and circulation.

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[1] For the convenience of those who may wish to consult the authors named, the references are subjoined as follows: Stokes on Diseases of the Heart and Aorta. W. King on "The Harmlessness of Adherent Pericardium," London Lancet, Nov., 1845. Article by Dr. Barlow in "Gulstonian Lectures." Dr. W. T. Gairdner on "The Favorable Terminations of Pericarditis, and especially

The foregoing conclusion, however, may be correct, and yet pericardial adhesions exert more or less agency in the development of cardiac disease in a certain number of cases. It is, therefore, an important object to determine the proportion of cases in which these adhesions are found to exist independently of other abnormal conditions of the heart. With reference to this point, Gairdner has analyzed 15 cases in which adhesions were found after death, the patients dying from various diseases. Of these cases, in 10 the heart was not enlarged, nor otherwise diseased. Of the remaining 5 cases, the heart was enlarged in all; but in 2 of the latter cases, valvular lesions coexisted; and in 2 the adhesions were not general, but partial. These 15 cases were collected by Gairdner from the records of 500 miscellane-

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in Adhesion of the Pericardium, with cases illustrating its Secondary Effects on the Heart," Edinburgh Monthly Journal of Medical Science, 1851. Bouillaud, in Leçons Cliniques sur les Maladies du Cœur, &c., Paris, 1853. The author last named states that he has met with more than fifty examples of pericardial adhesions in persons who had enjoyed good health for a long period, and who died with various affections foreign to the heart.

ous post-mortem examinations performed in the Edinburgh Infirmary, the cases of adherent pericardium only being selected in which the adhesions were so considerable and so situated as to restrain the movements of the heart. An analysis of 90 cases of adherent pericardium, collected from museums and catalogues by Henry Kennedy, of Dublin, yields results somewhat different from those obtained by Gairdner. Of these 90 cases, the heart remained healthy till death, in 34, and was enlarged in 51.[1] From this collection, all cases of valvular lesions were excluded. Of four cases of general adhesions disconnected from valvular disease, of which I have notes (and also the hearts, in my cabinet), in one only was the heart normal in other respects. In three cases there existed a moderate degree of enlargement.

In view of the discrepancy of the results of different analyses, the precise ratio of instances in which pericardial adhesions are found not to be accompanied by other abnormal conditions, which may be considered as consecutive, is not, as yet, determinable, but we are warranted in concluding that the proportion is at least one-third, excluding cases in which they are associated with valvular lesions.

It is evident that, in order to determine more fully the effects of adhesions on the heart, it is desirable to know, in the cases in which they are found not to be associated with any other abnormal condition, how long they have existed prior to death. Often this cannot be definitively ascertained. A collection of cases in which the length of time that had elapsed after the occurrence of pericarditis is known, and the heart examined after death, would be highly valuable with reference to the question under consideration.

Adhesions existing in connection with valvular lesions are properly excluded from the cases analyzed with a view to determine the effects of the former upon the heart. Valvular lesions, as is well known, in the great majority of cases, lead to cardiac enlargement, so that when these lesions coexist with adhesions, and the heart is found to be enlarged, it is fair to attribute the enlargement to the affection of the valves. And when valvular lesions and adhesions are combined, it may be concluded that the inflammatory affections giving rise to both occurred at the same time, inasmuch as clinical observation shows pericarditis in the majority of cases to be conjoined with endocarditis. It is also to be considered that the association of adhesions with enlargement of the heart (valvular lesions not being present) does not prove that the latter is an effect of the former. Other causes may have given rise to the enlargement, and the association may be merely a coincidence in some cases. Again, the enlargement may have proceeded not from the adhesions, but as a remote effect of the pericardial inflammation on the substance of the heart. The adhesions and the enlargement, in these cases, are coinciding effects of a common cause, viz., inflammation of the pericardium without any causative dependence on each other. Still farther, it is to be borne in mind that a moderate enlargement of the heart is not necessarily a serious affection. And, in point of fact, pericardial adhesions associated with a certain amount of enlargement, without the coexistence of valvular lesions, are often, if not generally, found after death in cases in which the symptoms had not denoted any cardiac affection, and death was owing to diseases having no apparent reference to the condition of the heart. In view of the several considerations just presented, it seems to be a logical conclusion that pericardial adhesions do not involve serious consequences to the extent which might be inferred from the statistics of Kennedy, showing that adhesions and enlargement are found after death to be associated in two-thirds of the cases examined, exclusive of the cases of valvular lesions. To these considerations it may be added that adhesions, enlargement, and valvular lesions combined, are sometimes borne for a long period. This fact is illustrated by the following case: A male patient, aged 68, was admitted into the hospital with advanced cardiac disease, and died the day after his admission. It was ascertained that 38 years had elapsed since an attack of acute rheumatism. The pericardium was found, on examination after death, to be universally and closely adherent by firm and evidently very old adhesions. The heart weighed 46 ounces. The enlargement was due mainly to hypertrophy and dilatation of the left ventricle and auricle, but the whole organ was increased in size. The curtains of the mitral valve were thickened, contracted, and the valve evidently insufficient. The aortic valve was sound.

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[1] Edinburgh Med. Journal, 1858.

The doctrine inculcated by Hope was, that pericardial adhesions invariably lead to enlargement of the heart. It has been seen that clinical facts abundantly disprove this doctrine. On the other hand, it has been recently maintained that the effect of adhesions is precisely the reverse of this; that they tend to produce atrophy of the organ. This view was first taken by Chevers.[1] It is adopted by Barlow and W. King in the papers already referred to. Professor Smith, of Dublin, thinks that he has found atrophy and hypertrophy to coexist with adhesions in about an equal proportion of cases. Stokes advocates this view on the ground of analogy with the apparent effects of pleuritic adhesions on the lungs, and of mechanical restraint on the voluntary muscles. Hope accounted for the production of hypertrophy on the principle that the heart, being mechanically restrained, was thereby excited to increased power of action to overcome the restraint; he applied, in other words, the principle on which valvular obstruction leads to hypertrophy. But the analogy does not hold good. As remarked by Stokes, "In adhesion, the normal condition of the muscle is interfered with and the contraction diminished; while in valvular obstruction, the muscle being free to act, increases in power, just as the voluntary muscles do when trained by exercise." Atrophy is supposed to be produced as an effect of pressure, which not only restrains the movements of the heart, somewhat like a bandage applied over the muscles of an extremity, but by interfering with the free supply of blood to the substance of the organ. This doctrine, however rational, with our present knowledge must be regarded as hypothetical; and, as a matter of observation, enlargement is much oftener found associated with pericardial adhesions than an abnormal diminution of the volume of the heart. Kennedy found evidence of atrophy in only five of the ninety cases analyzed by him. True atrophy, *i. e.*, in which the organ is reduced in volume, as is well known, is one of the rarest of cardiac lesions.

In conclusion, while it is not to be denied that pericardial adhesions do contribute to enlargement of the heart in a certain proportion of cases, nor that they may also, in rare instances, tend to an opposite result, they exist not infrequently for a long period without being followed by any appreciable morbid condition, and their tendency to the production of either organic change or functional disturbance is vastly less than was supposed by Hope. With this general view of the effects upon the heart and circulation, the remote consequences of pericarditis, so far as the pericardium is concerned, need not occasion much apprehension. It is not improbable that obliteration of the pericardial sac after acute pericarditis is, in some sense, a conservative result, preventing, in some instances, persistence of the inflammation in a chronic form. And it is highly probable that when pericarditis is followed by cardiac enlargement, exclusive of the cases in which valvular lesions coexist, the enlargement is due more to the condition of the muscular substance of the heart, produced directly by the inflammation, than to the effect of the adhesions. Assuming this to be the view most consistent with our present knowledge, its practical importance, as regards its bearing on the prognosis and management after recovery from pericarditis, is sufficiently obvious.

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[1] Guy's Hospital Reports, vol. vii.

Since the publication of the first edition of this work, I have noted ten cases in which the existence of old pericardial adhesions, by an organic attachment, was ascertained after death. Of these 10 cases, in two it is not stated whether the heart was, or was not, enlarged. In one case the heart was not enlarged; but it is not stated whether valvular lesions existed, or not. Of the remaining 7 cases, in 3 the heart was diminished in size, and in 1 case there was neither increase nor diminution. In all these 4 cases the valves were sound. In 3 cases there was more or less enlargement. Valvular lesions existed in each of these three cases.

In two of these specimens of old adhesions there was calcareous deposit beneath the pericardium. In one there were several calcareous plates; the largest, situated over the right ventricle, was four by three inches in diameter. In the other specimen the calcareous deposit near the base of the left ventricle extended through the muscular structure, leaving a portion exposed within the ventricular cavity.

How is the existence of pericardial adhesions to be ascertained during life?

If it be true that adhesions may remain innocuous for an indefinite period, and that they rarely, if ever, of themselves give rise to serious consequences, the importance of ascertaining their existence during life is less than it would be were their effects on the heart and circulation more important. Assuming the correctness of the general view of these effects which has been presented, I shall not dwell long on the present division of the subject.

Symptoms referable to the heart and circulation are not distinctive of adhesions. Whatever evidence is available must be derived from physical signs. The diagnostic points furnished by exploration require that the adhesions shall be nearly or quite universal, and they are more marked when, in addition to union of the pericardial surfaces, there exists firm adhesion of the pericardium to the parietes of the chest.

Percussion shows that the heart is in contact with the chest over a larger space than normal; in other words, the area of the superficial cardiac region is enlarged. But as this occurs whenever the heart is increased in volume, alone, it is not distinctive. Another sign obtained by percussion is significant. It is, persistence of the dulness within the same area in different positions of the body, and when the patient takes a deep inspiration. This shows that the heart is fixed, and that external adhesions prevent the lung from overlapping the organ, even when expanded by a forced effort.

Auscultation concurs with percussion in showing that the lungs do not extend over the heart on a full inspiration, provided the pericardium be united to the chest by pleuritic adhesions. This method of exploration furnishes no other diagnostic points. Clinical observation has not established any peculiar modifications of the heart-sounds. On theoretical ground, it is probable that the element of impulsion in the first sound is weakened, but the variations in this respect in health and disease are such as to render this alteration of slight import.

Palpation furnishes important signs. The apex-beat is frequently suppressed. It is not true that it is invariably wanting, even when universal and close adhesions exist. And, on the other hand, the apex-beat is not only suppressed in connection with different forms of disease, but it is not felt in all healthy persons. Alone, absence of the beat is by no means distinctive, but it is significant when taken in connection with other signs. Its suppression is accounted for by the fact that pericardial adhesions interfere with the projection and rotation of the heart's apex. When not suppressed, the beat may be felt higher than its normal position, namely, in the fourth instead of the fifth interspace, while the body is in a vertical position. This change of position, in connection with other signs, has considerable significance. If, however, the heart be much enlarged, the apex may be lowered, notwithstanding the adhesions.

Impulses may be felt in the intercostal spaces above the point of apex-beat, but this is not infrequently observed in cases of simple enlargement. Successive movements in different intercostal spaces, presenting an appearance of undulation, can hardly be considered as a sign of adhesions, if enlargement be present.

The apex-beat undergoes little or no alteration in its situation with change of posture. In health, the position of the beat is removed to the left, from half an inch to an inch, by changing the posture from that on the back to that on the left side. Pericardial and pleuritic adhesions prevent this lateral movement of the heart's apex. I have observed that the impulse not only remains in the same position, but retains the same force, the patient lying on the right side, as when lying on the back. In health, the impulse is either moved to the right by this change of posture, or, more frequently, lost. Another point pertaining to the apex-beat is its preserving the same position with the two acts of respiration. It is not depressed by a forcible inspiration, nor raised by a forcible expiration, to the same extent as in health.

A jogging or tumbling motion of the heart, as perceived by the hand, was considered by Hope as a distinctive sign. But violent and disturbed rhythmical action is not only observed in cases of enlargement and of merely functional disorder, but tranquil regularity of the heart's movements is perfectly compatible with universal and close adhesions. Nor is a sensation, communicated to the hand, as if the heart were restrained, or were struggling against an obstacle, of which Bouillaud speaks, to be relied upon.[1] Such a sensation must involve a preconceived idea that adhesions exist.

Inspection sometimes furnishes signs which are distinctive, namely, retraction of the intercostal spaces, and depression of the epigastrium to the left of the xiphoid cartilage, occurring synchronously with the ventricular systole. The depression movement of the epigastrium is due to the attachment of the base of the pericardium to the cordiform tendon of the diaphragm; the retraction movement of the intercostal spaces is caused by the drawing-in of the spaces when the ventricles contract, and is most marked when the pericardium is attached to the parietes of the chest. One or two, and possibly three, of the intercostal spaces may present retraction, and in some instances the ribs are also retracted. Depression of the epigastrium may be present alone, or associated with intercostal retraction. In some instances, the xiphoid cartilage, and even the lower portion of the sternum, is drawn inward, apparently with considerable force. These signs, when present in a marked degree, and especially in combination, are highly distinctive.

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[1] "On sent à la main que le jeu du cœur est embarrassé, difficile, &c." Leçons Cliniques, 1853.

To recapitulate the physical signs denoting pericardial adhesions, they are as follows: the area of præcordial dulness on percussion remaining unaltered in different positions of the body, and not affected by a deep inspiration; the limits of the respiratory murmur not affected by a deep inspiration; the apex-beat often suppressed, and, if not suppressed, often raised above its normal position; the apex-beat, if felt, unaffected, or affected but slightly, by changes of posture from the back to either side, and by forcible inspiration or expiration; retraction of one or more intercostal spaces, together with the ribs in some cases, and depression of the epigastrium synchronously with the ventricular systole.

These signs strengthen each other by combination. The more these are combined, the greater the significance of each. Not one, however, is constant, and all may be wanting in cases of pericardial adhesions. They are not marked unless the pericardium be attached to the thoracic walls by pleuritic adhesions, and these do not always coexist with union of the pericardial surfaces. A positive diagnosis, therefore, is only practicable in a certain proportion of cases. If it be known that a patient has had, at some past period, an attack of pericarditis, this fact renders less physical evidence necessary for the diagnosis than if the previous history contained no information with respect to that point. The fact of the patient having had acute rheumatism adds weight to the conclusion drawn from the physical signs. Contraction of the chest, limited to the præcordia, is also a collateral point of evidence, as showing that pericarditis has existed.

CHAPTER VIII.

INFLAMMATORY AFFECTIONS OF THE HEART. ENDOCARDITIS—MYOCARDITIS.

Endocarditis—Definition—Anatomical characters—Pathological relations and causation—Symptoms—Physical signs—Diagnosis—Prognosis—Treatment. Myocarditis.

ENDOCARDITIS.

INFLAMMATION of the endocardium, the membrane which lines the cavities of the heart and is duplicated to cover the valves, is called endocarditis. This name originated with Bouillaud, who was the first to recognize clearly the occurrence of inflammation in this situation. Clinical researches have shown that this disease, the nosological existence of which dates from less than half a century ago, is by no means infrequent. It occurs as a complication of acute rheumatism in a considerable proportion of cases. The knowledge of its frequent coexistence with this affection is one of the most important of the developments of modern medicine. The remote effects of endocarditis, as involved in the valvular lesions which have been considered in a former chapter, invest the disease with much importance. Inflammation here, as in other situations, may be acute, subacute, and chronic; but it is hardly practicable to make these distinctions, clinically, and hence it suffices to consider the subject under the head, simply, of endocarditis. In the consideration of this subject, the same divisions will be adopted as in treating of acute pericarditis, namely, the anatomical characters of the disease, its pathological relations and causation, its symptomatic phenomena, its physical signs, the diagnosis, the prognosis, and the treatment. These divisions will be taken up in the order in which they have just been named.

ANATOMICAL CHARACTERS OF ENDOCARDITIS.

Endocarditis is limited, in the vast majority of cases, to the membrane lining the cavities of the left side of the heart. The lining membrane of the right auricle and ventricle is rarely inflamed. When inflammation does exist in the right side, it is also present in the left side. All portions of the endocardial membrane within the left cavities of the heart are not equally subject to inflammation. The portions covering the valves and lining the orifices are especially prone to become inflamed. Endocarditis is generally limited to these situations. The membrane here is most exposed to the action of the blood-currents; the valvular portion is in constant motion, and considerable tension or stretching must take place with each ventricular systole. But another, and perhaps a stronger, reason for the limitation of inflammation to these situations is derived from the fact that the membrane is here underlaid by fibrous tissue, while in other portions it is in close proximity to the muscular walls of the heart. The rule as regards the liability of the left side of the heart to endocardial inflammation is applicable after birth, but it does not hold good during intra-uterine life. There are grounds for the belief that the fœtus *in utero* is subject to endocarditis, and that at this period the inflammation is seated in the right side of the heart. The malformations which have been considered in a previous chapter are in a measure thus accounted for.

Opportunities for inspecting the morbid appearances during the progress of endocarditis are not often presented. The disease very rarely proves fatal. It is not the immediate danger, but the remote consequences, which render it a serious affection. The anatomical characters observed in the occasional instances in which death has occurred when inflammation existed, embrace here, as in other situations, redness from vascular injection, alterations in the membrane itself, and the presence of inflammatory products.

Redness due to endocarditis is caused by injection of the vessels which ramify in the areolar tissue beneath the membrane. It is not always found when inflammation undoubtedly existed at the time of death. It may disappear as a post-mortem change. On the other hand, mere redness is by no means adequate evidence of the existence of inflammation. It is often observed in the cavities of the heart, as well as in the large vessels, as an effect of the imbibition of hæmatin dissolved out of the red globules of the blood which these cavities and vessels contain after death. Under these circumstances, it is a cadaveric staining of the membrane, and is an effect of the decomposition of the blood. It is observed frequently in post-mortem examinations, more especially when these are made two or three days after death, or when the warmth of the weather favors putrefactive changes; and it is a post-mortem condition found after certain diseases in which the blood undergoes notable changes prior to death. The redness from imbibition is distinguished from that due to inflammation, by the following points of difference: It is not an arborescent, but a uniform redness, and when examined with a lens, injected vessels are not visible. It has a deeper or darker color than inflammatory redness. It is more likely to be observed in the right than in the left side of the heart, a larger quantity of blood usually remaining in the right cavities after death. It is not limited to the valves or orifices, and usually extends into the arteries, where it is more conspicuous than in the cardiac cavities. The redness is most marked in the dependent parts of the heart and vessels. Exclusive of the discoloration, the membrane preserves its natural appearance, it is firm and polished as in its normal condition, and does not present any of the inflammatory products. Whatever may be the characters pertaining to redness, if the membrane be normal in all other respects, and the products of inflammation wanting, the evidence of the inflammatory state is insufficient.

Anatomical changes in the membrane are much more distinctive of inflammation than redness. These changes are, loss of the transparent, smooth, polished appearance which the membrane has in a healthy state, instead of which it becomes opaque, rough, velvety, and felt-like; more or less swelling and softening; brittleness of the subjacent areolar tissue, in consequence of which it is more easily detached than in its normal condition. Anatomical changes due to ancient inflammation or to atheromatous deposit and hypertrophy of the endocardium, are, of course, to be distinguished from those which denote endocarditis existing at the time of death.

Other characters relate to products of inflammation. As regards these, the endocardium, although resembling serous membranes, differs from the latter in not being a shut sac, within which inflammatory products are collected and retained for a greater or less period. Morbid products are liable to be detached, washed away by the currents of blood, and carried along with the circulation. The endocardium differs from serous membranes in another important point, namely, its free surface is in contact with the blood itself; and while this fluid, in motion, detaches and removes morbid products, it may also furnish deposits by yielding a portion of its fibrin which undergoes coagulation.

Products of inflammation in endocarditis may be derived from two sources, namely, the coagulation of fibrin, just alluded to, and the exudation of lymph occurring here as in serous inflammations. In the one case they are derived from the blood within the cavities of the heart, and in the other case, from the blood in the vessels situated in the areolar tissue beneath the endocardium. It has been supposed. that exudation of lymph from the blood contained within the vessels, takes place here, as in analogous structures when inflamed, and that it occurs on the free surface of, and beneath the endocardium. If not detached and washed away by the blood-currents, lymph exuded on the free surface remains attached to the membrane, forming membranous-like layers, as in cases of pericarditis or pleuritis, but not in the same abundance. The roughness produced by the exudation of lymph or the alterations of the membrane, attracts, as it were, the fibrin from the blood, and leads to its precipitation in a coagulated state. The lymph is like a foreign substance, and becomes coated with fibrin, like the threads passed through arteries in Dr. Simon's well-known experiments. An increased proportion of fibrin in the blood, which characterizes in a marked degree acute rheumatism, probably favors the deposit from the latter source. This twofold origin of the products of inflammation is important in its bearing on the treatment of endocarditis.

In addition to these inflammatory products, morbid growths are to be reckoned among the anatomical characters of endocarditis. These either enter into, or constitute, what are commonly known as vegetations. The so-called vegetations are found especially either at the base or the free extremities of the valves. They have frequently the form of small granular masses or beads, varying in size from that of a pin's head to a millet seed, studding the margins of the curtains of the mitral valve, and fringing the crescentic extremity of the fibrous portion of the segments of the valves of the aorta. The true vegetations are outgrowths from the membrane, attributable, according to the doctrine of Virchow, to proliferation of the connective tissue which enters into the structure of the endocardium.

The products of inflammation before mentioned, namely, exuded lymph and deposited fibrin, are found especially in the same situations as the morbid growths, that is, on the membrane of the valves and orifices of the left side of the heart. They may be presented, as already stated, in thin membraniform layers adherent by agglutination, rendering the membrane opaque and apparently thickened, or they may be disposed in granular or bead-like masses resembling in appearance the vegetations just described. They are more easily detached than the true vegetations, that is, the outgrowths, and do not present, on microscopical examination, the characters which distinguish the latter. Frequently, however, the true vegetations are increased in size by the deposit upon them of coagulated fibrin. Thus increased, they may form excrescences of the size of a pea, and they are sometimes even considerably larger than this. The nuclei of these excrescences may be either morbid growths, that is, true vegetations, or exuded fibrin, or both, to which more or less coagulated fibrin has been added. Of the different elements which enter into the excrescences, two only are exclusively inflammatory, namely, exuded lymph and the morbid growths. The deposit of fibrin from the blood in the heart-cavities occurs as a result of endocarditis, but it may also occur whenever the membrane is roughened, more especially if the fibrinous constituent of the blood is abnormally increased. In so far as excrescences consist of coagulated fibrin they are liable to be formed in cases of valvular lesions although endocarditis may not exist. Fibrinous deposits are most apt to occur on the valvular surfaces which are exposed to the direct currents of blood, namely, on the auricular aspect of the mitral, and on the ventricular aspect of the aortic valves. It is to be added, that Virchow denies the occurrence of the exudation of lymph in endocarditis, accounting for the formation of the excrescences and membraniform layers wholly by proliferation or morbid growth and the deposit of coagulated fibrin from the blood within the cavities of the heart.

Other morbid changes which may occur during the progress of endocarditis are, loss of substance, or destruction of portions of the membrane, by ulceration and erosion; perforation of the valves; lacerations, and, according to Bouillaud, occasionally gangrene. These are rare occurrences as immediate results of the inflammatory processes. Lacerations and erosions are the least infrequent. Adhesion of the valves to each other, to the walls of the heart, or, of the semilunar valves to the inner surface of the artery, are to be included among the comparatively rare anatomical changes incident to recent endocarditis.

The opportunity of inspecting the morbid appearances during the different stages of the progress of endocarditis, as already stated, is rarely presented, inasmuch as the disease seldom proves fatal. For this reason the appearances found after death in the experiments made by Richardson, in which endocarditis was artificially induced in inferior animals by the introduction of lactic acid into the blood, are of much interest and value.[1] In sixteen experiments on dogs, cats, and rabbits, endocarditis was invariably produced when a certain quantity of lactic acid, largely diluted, was injected into the peritoneal cavity. If the animals died or were killed at a period when the symptoms denoted commencing inflammation, the endocardial membrane presented a brilliant vermilion color; it had a velvety or villous appearance, and beads of lymph or fibrin were abundant. At a somewhat later, but still early, period the auriculo-ventricular valve became thickened and œdematous. The writer says: "I have seen the segments of the tricuspid valve fixed in this swollen condition, resembling each an injected uvula, and lying so close to each other that, when the heart was contracting, they must have cushioned against each other, thus fulfilling their office of preventing regurgitation passively, *i. e.*, without tension or movement. In this œdematous stage, if the valve be pricked with a needle, a clear white lymphy fluid exudes, and by frequent pricking, the valve structure, emptied of its effusion, collapses and assumes a flaccid condition." At a later period "the valves remained thickened, but the red color and œdematous state were both reduced. Beneath the endocardial surface of the valve there was a paleness as from coagulated effused lymph. If the needle be applied now, there is no exudation; the valve has some limited play, unless it is bound down by adhesions and its structure is firm.

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[1] An Experimental Inquiry on Endocarditis, by the Synthetical Method. By Benjamin W. Richardson, M.D., &c. See appendix to Prize Essay, London, 1853.

Beads which generally fringe the margin of the valves all around, from being œdematous prominences in the earlier stages, are pearly looking, and are moderately firm." Still later, the writer describes the valves as shrunken, having regained imperfect play, but still thickened and unyielding. It is probable that this account of the morbid appearances in inferior animals may be applied analogically to endocarditis in man, during periods of the disease when the anatomical characters can be studied in only the very rare instances in which a fatal result takes place, and when a sufficient time has not elapsed after death for important post-mortem changes to have ensued. These experiments will be again referred to in connection with the causation of endocarditis.

A point observed by Richardson with regard to the situation of the anatomical changes in artifically induced endocarditis may be here mentioned. These changes were mostly confined to the auriculo-ventricular valves. He states: "A very slight thickening, not sufficient at any time to interfere seriously with their duties, is all I have ever observed in the semilunar valves on either side."

The remote effects of endocarditis are vastly more serious than the immediate anatomical changes. The latter lay the foundation of the valvular lesions which were considered in Chapter III. These lesions are slowly induced in consequence of the morbid products and growths which inflammation induces, their progressive increase from the deposit of fibrin, the process of calcification, &c., together with the softening, friability, and sometimes solution of continuity of the endocardium and the subjacent tissue. Insufficiency of the valves and contraction of the orifices are ulterior consequences, giving rise to enlargement of the heart and other pathological effects which have been treated of in preceding chapters. With reference to their eventuation in valvular lesions involving either obstruction or regurgitation, or both, the immediate anatomical changes in endocarditis are of great importance. In a very large proportion of cases, valvular lesions owe their origin to these changes. Hence, a person attacked with endocarditis is liable, at a period more or less distant, perhaps after many months or years, to fall a victim to organic disease of the heart. The rapidity and extent of the remote effects will be proportionate, other things being equal, to the amount of changes which remain after the endocarditis has ceased. In this respect different cases doubtless vary. It is possible for the fibrin or lymph which adheres to the free surface of the membrane to be gradually washed away by the blood-currents, for the inflammatory products beneath the endocardium to be removed by absorption, and the normal condition of the valves to be restored, so that no ulterior evils follow the disease at any period, however remote. This happy termination is an exception to the general rule.

PATHOLOGICAL RELATIONS AND CAUSATION OF ENDOCARDITIS.

Endocarditis resembles pericarditis in the infrequency of its occurrence as an idiopathic affection. It occurs, however, independently of other affections, oftener perhaps than is generally supposed, its latency as regards symptoms being such that it is overlooked. In the great majority of the cases in which its existence is ascertained, it is associated with acute articular rheumatism. It is comparatively a very rare affection as occurring in other pathological connections. Rheumatic endocarditis is sufficiently common for cases to fall frequently under the observation of the medical practitioner. Endocarditis and pericarditis are not very infrequently associated in cases of rheumatism. The combined affections are designated *endo-pericarditis*. Rheumatic pericarditis very rarely exists without endocarditis. But the converse of this statement does not hold good; endocarditis often exists without being associated with pericarditis. With reference to the frequency of endocarditis in acute rheumatism, and the relative proportion of cases in which endocarditis, pericarditis, and endo-pericarditis, respectively, occur in the cases of rheumatism with cardiac complication, the following statistics may be cited: Of 474 cases collected from various sources and analyzed by Fuller, endocarditis existed in 214, the ratio being as 1 to every 2.25 cases. These cases were reported by four different observers, and the analysis of each collection gives not far from the same ratio as when they are analyzed collectively. Of 204 cases of rheumatism with cardiac complication of some kind, endocarditis existed in 138, pericarditis in 19, and endo-pericarditis in 38. Bamberger, from his statistical researches, reckons the proportion of the cases of rheumatism in which endocarditis occurs, at 20 per cent.[1] The accuracy of statistics is, of course, based on the practicability of determining the coexistence of endocarditis in cases of rheumatism, and there are certain liabilities to error which will be noticed in connection with the subject of the diagnosis. Suffice it to say here that there is reason to suppose that in more or less of the cases analyzed by Fuller, the diagnosis of endocarditis was based on insufficient evidence; in other words, the frequency of the occurrence of endocarditis in rheumatism is exaggerated in the statistics from his work.

The portion of the endocardial membrane which covers the mitral valve is affected oftener and to a greater extent than the portion which is in relation with the aortic valves, in cases of rheumatic endocarditis. This fact is shown by the signs during life which indicate the mitral valve as the seat of disease, and by the larger proportion of cases in which mitral valvular lesions are observed when organic affections of the heart are traceable to an attack of rheumatism. The anatomical characters of endocarditis artificially induced in inferior animals, in Richardson's experiments, as has been stated, were mostly limited to the auriculo-ventricular valves.

What is the nature of the pathological relation existing between endocarditis and acute rheumatism? The remarks in connection with a similar question as applied to pericarditis are here equally applicable. The endocardial inflammation is not developed as a metastasis of the articular affection. The former proceeds from the same morbid condition which determines the latter. Both are effects of a common internal cause. The affection of the joints is not lessened by the occurrence of endocarditis. Nor does the affection of the joints always precede the development of the endocardial inflammation. The latter occasionally takes precedence. Clinical observation shows that the liability to endocarditis is in proportion to the acuteness of the rheumatism; yet it is to be borne in mind that the mildest cases of rheumatism are not exempt from this liability. Endocarditis may be developed at any period during the career of acute rheumatism; but the statistics of Fuller go to show that the liability is greatest between the sixth and twentieth days of the disease. The influence of youth in the development of rheumatic endocarditis does not appear to be so marked as with respect to pericarditis.

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[1] *Vide* Niemeyer.

Endocarditis, as well as pericarditis, is one of the secondary inflammations liable to become developed in connection with renal disease. Of 39 fatal cases of recent endocarditis analyzed with reference to this point by Dr. T. K. Chambers,[1] it was referable to disease of the kidneys in 12. In the same collection of cases, the affection was connected with acute rheumatism in only 9. These figures show a larger proportion of *fatal* cases from uræmic than from rheumatic endocarditis. The same, it has been seen, is also true with regard to pericarditis. The fact is owing to the greater fatality of endocarditis, as well as pericarditis, when associated with renal disease. The latter affection, existing to an extent to induce inflammation of any of the important organs of the body, generally proves fatal. On the other hand, acute rheumatism complicated with cardiac disease, very rarely ends fatally. The difference in fatality between the two affections thus accounts for the preponderance of fatal cases of endocarditis with renal disease, while the proportion of instances in which endocarditis is associated with acute rheumatism is vastly greater.

As regards the pathological relation existing between endocarditis and disease of the kidneys, the remarks made with respect to pericarditis are equally applicable. It is most consistent with our present knowledge to attribute the development of the former, as well as the latter, to the accumulation of urinary principles in the blood. The analogy of structure between the endocardium and serous membranes explains the liability of the former to become inflamed under the same conditions which occasion inflammation of the latter.

It is stated that in non-rheumatic endocarditis, the aortic valves are more likely to be the seat of inflammation than the mitral, the reverse being true, as has been seen, of rheumatic endocarditis.[2] But in a pretty large proportion of instances, the endocardium in both situations is affected, whatever may be the pathological connection of the disease.

Endocarditis and pericarditis, as already stated, are frequently associated. This combination existed in 38 of the 204 cases analyzed by Fuller. Clinical observation shows that either affection may take precedence of the other in point of time. Pericarditis, in fact, rarely exists without the coexistence of endocarditis. Hence, it may seem reasonable to infer that inflammation in either situation tends to develop it in the other, and, especially, that pericarditis leads to the development of endocarditis. It is doubtful, however, whether any causative relation exists between the two affections. When associated, they are probably effects of a common pathological condition. This condition, in the great majority of cases, is either rheumatic or uræmic poisoning; but much oftener the former than the latter, clinical experience showing that endo-pericarditis is more frequently associated with rheumatism than with disease of the kidneys.

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[1] Op. cit.

[2] Bellingham, op. cit., part ii, p. 348.

Endocarditis is sometimes associated with inflammation of the pulmonary structures—pleurisy or pneumonia. It is less frequently associated with these affections than pericarditis. The difference, as regards rheumatic endocarditis and pericarditis, is shown by the following statistics by Fuller:[1] Pulmonary inflammation, of some kind, existed in only 8 of 80 cases (a ratio of 1 to 10) of acute rheumatism complicated with endocarditis. It existed in 7 of 12 cases (1 to 1.7) of acute rheumatism complicated with pericarditis; and in 19 of 27 cases (1 to 1.4) in which endo-pericarditis was a complication. Pulmonary inflammation, however, exists oftener in cases of rheumatism complicated with endocarditis, than in rheumatic cases devoid of any cardiac complication. Thus Fuller found it to occur in only 7 of 127 cases in which rheumatism was uncomplicated with disease of the heart. This fact may seem to show that pulmonary inflammation exerts some influence, although feeble, in determining the occurrence of endocarditis. It is, however, more rational to conclude, in view of all the facts, that there does not exist any pathological relation between pneumonia or pleurisy and endocarditis when they are associated, but that both may proceed from a common cause. The occurrence of pulmonary inflammation in a proportion of cases somewhat larger when rheumatism is complicated with endocarditis, than when this complication does not exist, may arise from a greater intensity of the blood-poisoning. If any pathological relation exist, it is perhaps as rational to suppose that the pulmonary inflammation is dependent on the cardiac affection, as that the former is involved in the causation of the latter.

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[1] Op. cit.

Endocarditis is occasionally developed in connection with the eruptive and continued fevers, and with the morbid condition considered as pyæmia. Its occurrence in these connections is rare. It may also be produced by injuries of the chest; but cases of traumatic endocarditis are extremely infrequent.

It is evident from the foregoing account of the pathological relations of endocarditis, that, exclusive of its connection with acute rheumatism and Bright's diseases, it is, practically, not of much importance, since it so seldom comes under the observation of the medical practitioner. But, as before remarked, its occurrence as an idiopathic affection, especially in early life, may not be quite as infrequent as is generally supposed. The number of cases of valvular lesions in those who have not had acute rheumatism, is a ground for suspecting that it occurs oftener than it is recognized. As a complication of rheumatism and renal disease, it possesses very great importance, in view of the frequency of its occurrence, and its remote evils. Its importance, in a certain point of view, is greater as a complication of rheumatism than of disease of the kidneys, for in the latter association it is generally combined with pericarditis, and it is developed under circumstances which offer small encouragement to expect recovery. On the other hand, in connection with rheumatism, the immediate danger is slight, and there is ground for hope that remote evils may not occur.

The experiments of Richardson, to which reference has been made, are of interest and value in their bearing on the causation of endocarditis. After injecting into the peritoneal cavity of the dog a solution of lactic acid containing ten per cent. of the acid (an operation almost painless) the liquid is soon absorbed, and in about twelve hours the symptoms and physical signs denote the development of endocarditis. The morbid appearances observed in different stages of the endocardial inflammation have been stated. These appearances are mostly confined to the right side of the heart, and are especially seated in the tricuspid valve and orifice. Richardson attributes the inflammation to the local action of the lactic acid, which he supposes to act on the right side of the heart, because, being absorbed by the veins, "it comes into contact with the inner surface of the right side of the heart first; in the pulmonic circuit, it undergoes some loss, and so entering the left cavity is less active in its effects. In other words, in so far as the heart is concerned, the poison is derived from the systemic circuit, and is lost in the pulmonic circuit." He regards his experiments as proving, synthetically, that rheumatic endocarditis is produced by a similar agent. Analysis furnishes corroborative evidence by showing the acidity of the excreta in acute rheumatism, especially the perspiration. But in rheumatism the endocarditis is seated in the left, not in the right cavities of the heart. To account for this Richardson supposes that the poison in rheumatic endocarditis is a product of respiration, and is contained in the arterial blood. "Hence, it comes in contact, first, with the inner surface of the left side of the heart; while, in the systemic circuit, it undergoes loss or combination, so that the blood returning by the veins is not poisoned, and the right side of the heart escapes." That the inflammation is produced by the direct contact of a poisonous agent, when artificially induced, and in rheumatic endocarditis, Richardson considers as proved by the limitation of the inflammation to one side of the heart, for if a blood-poison were to produce its effect through the nutritive vessels of the part, it would seem that the two sides of the heart should be equally affected, inasmuch as both are supplied from a common source with the same blood. The fact that during fœtal life, when the lungs do not fulfil the office of respiration, the right side of the heart is liable to endocarditis corroborates the views of Richardson.[1]

Endocarditis may give rise to immediate pathological results which are important. Among these are to be reckoned emboli, or plugs, consisting of detached vegetations or excrescences, which, propelled with the current of blood into the arteries, are at length arrested in their course in trunks too small to permit their farther progress, giving rise to arterial obstruction and diminished supply of blood to certain parts. This subject has already been considered in connection with valvular lesions,

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[1] It is proper to state that the conclusion of Richardson with respect to the production of endocarditis by the injection of lactic acid has been called in question. A German experimentalist, Dr. G. Reyher, asserts that the same appearances are often found when apparently healthy dogs are speedily killed by prussic acid, or blowing air into the jugular veins. *Vide* Brit. & For. Med.-Chir. Rev., Jan., 1862, from Virchow's Archiv. Also, Amer. Journ. of Med. Sciences, April, 1862.

Chapter III, to which the reader is referred.[1] It is sufficient to say that the production of emboli is an accidental event which may occur during the progress of endocarditis, as well as afterward when lesions of the valves have taken place as a remote effect of endocardial inflammation. Their occurrence, however, is more frequent in connection with valvular lesions. The phenomena which are symptomatic of embolism of the cerebral arteries, such as apoplectic seizures, paralysis, &c., rarely enter into the clinical history of cases of endocarditis. But the liability to their occurrence should be borne in mind.

The solidified products in cases of endocarditis, namely, fibrin and lymph, are, to a greater or less extent, disintegrated by the blood-currents and carried into the circulation, either in solution or suspended in the form of minute particles. It is supposed that the comminuted solid deposits, transported to different organs, and becoming arrested in the capillary vessels, may give rise to vascular obstruction and secondary inflammation in these organs. The kidneys and spleen are most likely to be the seat of disease thus induced. These effects are primarily mechanical; but it is highly probable that morbid changes in the blood itself are sometimes induced by the admixture of the liquid products of endocardial inflammation. It can hardly be otherwise, if, as is not improbable, purulent matter is occasionally formed on excoriated or ulcerated surfaces, which are in some instances observed after death in cases of endocarditis. Our knowledge of these effects, as derived from clinical facts, however, is as yet too meagre to warrant any important conclusions. The fact that rheumatic endocarditis very rarely ends fatally, and rarely presents symptoms which denote purulent infection of the blood, goes to show the benignity, in most instances, of the products of endocarditis.

The formation of large masses of fibrinous coagula in the cavities of the heart belongs among the immediate pathological effects of endocarditis. The contact of the blood with the inflamed membrane and the commingling of the liquid products of endocardial inflammation have been supposed to induce coagulation, giving rise to the ante-mortem clots which were called by the older writers polypi of the heart. As an effect purely of endocarditis, this must be extremely rare, in view of the fact already repeatedly stated, namely, that endocarditis is fatal in but a very small proportion of cases. It is probable that when this event does occur during the progress of endocarditis, other conditions are involved which are more concerned in the occurrence of the event than the endocardial inflammation, such as cardiac enlargement, weakness of the heart from any cause, and a state of the blood which renders it prone to coagulation. Clinical observation fails in furnishing evidence of the formation of coagula during the progress of endocarditis as often as the writings of Bouillaud and some others would lead the observer to expect; but that a certain amount of agency in the production of this event is derived from the disease, is not to be denied.

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[1] *Vide* page 168.

SYMPTOMS OF ENDOCARDITIS.

The symptoms of endocarditis are less distinctive even than those of pericarditis. Occurring, generally, in connection with acute rheumatism, its symptomatic phenomena are merged in those of the latter affection. Hence, although its occurrence as a complication of rheumatism is so frequent, even the existence of such a disease has been known only within a late period. In a large proportion of cases there are no symptoms which attract attention to the heart as the seat of any disease. Examination, however, with a view to determine the presence, or otherwise, of phenomena which point to endocarditis, may elicit symptoms which are of importance in the diagnosis. These symptoms consist of pain referable to the heart, symptomatic fever, and excited action of the organ, or palpitation. Symptoms arising from obstruction to the passage of blood through the orifices of the heart do not belong, properly, to the symptomatology of endocarditis, but are due either to lesions resulting from endocardial inflammation, or to accidental events, such as the formation of coagula. These symptoms have been considered in preceding chapters.

Pain is very rarely a prominent symptom, and is often wanting. When present, it is not easy to refer it to the endocardium, except by taking into account other symptoms and the physical signs. The pain is not acute or lancinating, but dull or obtuse. A sense of uneasiness, hardly amounting to pain, is sometimes referred to the præcordia. The suffering from the affection of the joints is usually so much greater than the pain arising from rheumatic endocarditis, that the patient will not be likely to speak of the latter until interrogated with respect to it. If præcordial pain be marked in cases of rheumatism, there is reason to suspect, not alone endocarditis, but pericarditis or pleurisy, and these affections are to be excluded by the absence of other symptoms and signs before concluding that the pain is due exclusively to endocardial inflammation.

It is probable that inflammation of the endocardium alone would generally give rise to more or less febrile movement. But in cases of rheumatic endocarditis it is difficult to say how much of the febrile movement is symptomatic of the cardiac inflammation. Taken alone, this symptom is in no wise distinctive. If febrile movement be suddenly developed or increased when it is not referable to a fresh attack of any of the joints, or to inflammation seated elsewhere, it is fair to attribute it to the occurrence of endocarditis, if other symptoms and signs indicate the existence of this affection. Febrile movement, under the circumstances just stated, should excite suspicion of the occurrence of endocarditis, and lead to examination with reference to other symptoms and to signs.

Endocardial inflammation may excite the muscular action of the heart, inducing a species of palpitation. Of this the patient may be conscious and make complaint; and it is apparent to the hand placed over the præcordia, and also by the pulse. The action of the heart may be irregular, as well as unduly excited. The force of the pulse is observed in some instances not to correspond with the activity of the heart, as shown by the impulse felt in the præcordia. These symptoms, occurring in the course of acute rheumatism, should lead to the suspicion of cardiac disease, which may prove to be endocarditis. Like the other symptoms, these alone are of little value as distinctive of the disease, but, taken in connection with other symptoms and with signs, they have considerable significance.

The symptoms which have been mentioned derive their importance, as indicative of endocarditis, chiefly from their occurrence in the course of acute rheumatism. The clinical history of idiopathic endocarditis, based on analyses of recorded cases of the disease, is yet to be written. The information obtained by physical exploration is more important, and to this, attention will now be directed.

PHYSICAL SIGNS OF ENDOCARDITIS.

Increased extent and degree of dulness on percussion, due to tumefaction of the heart and accumulation of blood within its cavities, is considered by Bouillaud and others as a physical sign of endocarditis. Assuming that these conditions are incident to the disease, it may be doubted whether the cardiac enlargement often, if ever, much exceeds the limit of healthy variations; and if the size of the heart be found to be abnormal, it is impossible to say that it is owing to an existing endocarditis, unless it have been ascertained by previous examinations that, prior to the present attack, there was no enlargement of the heart. If, by successive explorations from day to day, it be ascertained that the heart becomes enlarged, as it were, under the eyes of the observer, and it be clear that pericarditis does not coexist, the increased size may be attributed to endocardial inflammation, provided other signs and the symptoms are sufficient for the diagnosis. How far the size of the heart undergoes alterations during the progress of endocarditis I am unable to say from my own observations; but it is evident that percussion cannot afford very important information with reference to the diagnosis of this disease, except in a negative point of view, that is, by aiding in the exclusion of other cardiac affections, more especially pericarditis. In this point of view it is of much importance.

Palpation and inspection furnish evidence of excited action of the heart. The impulse is seen and felt to be more violent than in health, or out of proportion to the amount of febrile movement which exists. These signs, however, are present in but a certain proportion of cases, and they may continue only during the early part of the disease. Moreover, abnormal activity of the heart, giving rise to increased force and extent of impulse, is sufficiently common in cases of functional disorder, irrespective of endocarditis. The signs furnished by these methods of exploration, are, therefore, of little value except as associated with other evidence of endocardial inflammation.

The only positive proof of the existence of endocarditis is derived from auscultation, and consists in the development of an endocardial murmur. Clinical experience has established the fact that a murmur accompanies inflammation of the endocardium. This murmur is usually soft, having the character of a bellows-sound, and is systolic, that is, it accompanies the first or systolic sound of the heart. It is not, however, developed always, and perhaps but rarely, at the commencement of the inflammation. A certain period elapses before this sign is discovered, and this period probably varies in different cases. It is not easy to determine the interval, since the existence of endocarditis cannot be recognized prior to the production of a bellows-murmur. An approximation to correctness of observation with respect to this point is obtained by ascertaining the duration of the symptoms which point to cardiac disease, anterior to the development of the murmur. The average duration of inflammation before murmur occurs, is yet to be determined. Richardson found that a murmur invariably followed the symptoms of endocarditis artificially induced in inferior animals, the interval varying in his different experiments.

It is needless to remark that the presence of an endocardial murmur is not, in itself, evidence of existing endocarditis. Murmur occurs in connection with valvular lesions which have taken place as remote effects of inflammation, or as results of other causes. It occurs in consequence of blood-changes, independently of an inflammatory affection or any organic disease of the heart. The inquiry then arises, what are the circumstances which, taken in connection with the presence of a murmur, render it a diagnostic criterion of endocarditis?

The development of a murmur in the course of acute rheumatism, in conjunction with symptoms denoting cardiac inflammation, renders it almost certain that endocarditis has occurred. If, after the lapse of several days, an endocardial murmur be detected, which previous explorations, made with sufficient care, have failed to discover, the practitioner should conclude that it is a sign of endocarditis. This conclusion is rendered more positive, if increased febrile movement, excited action of the heart, or pain in the præcordia, are observed to precede or accompany the development of the murmur. But it will happen not infrequently that a murmur is present when the patient first comes under observation. This is the case especially in hospital practice, patients being admitted after rheumatism has continued already for a greater or less period. It happens also in private practice, since endocarditis may occur at the very commencement of an attack of rheumatism, and may even precede it. The difficulty, in these instances, is to determine that the murmur has been recently developed. It may have existed prior to the attack of rheumatism, being dependent on some organic mischief, or on inorganic morbid conditions. There is strong ground for suspecting that the murmur pre-existed, if the patient have had rheumatism before. This difficulty is sometimes insuperable; but with reference to it, several points are to be considered. A murmur due to endocarditis is generally referable to the mitral orifice; in other words, it is either limited to, or heard with greatest intensity over, or near the point of the apex-beat of the heart. It has been seen that in rheumatic endocarditis, the inflammation is seated especially at the mitral valve, and clinical observation shows that, in most instances, the murmur emanates from this situation. The fact of the murmur being mitral, shows that it is not inorganic, since an inorganic murmur, in the vast majority of cases, if not invariably, is produced at the arterial orifices. If, however, the murmur in question be aortic, other circumstances are to be taken into account in determining that it is not inorganic. These circumstances have been considered in a preceding chapter.[1] Having determined that the murmur is not organic, the question then is, whether it be due to valvular lesions which have existed for a greater or less period, or whether it denote an existing endocarditis. Valvular lesions lead to enlargement of the heart. Now, if the heart be found to be enlarged, it is probable that the murmur proceeds from valvular lesions. Endocarditis, it is true, may occur, and is perhaps more likely to occur, in cases of rheumatism, when the heart is already affected with organic disease, but, under these circumstances, the murmur cannot be considered as diagnostic of endocarditis. On the other hand, if the heart be not enlarged, the chances are in favor of the murmur being due to endocarditis, especially if the symptoms render the existence of the latter probable. Another point relates to the murmur itself, assuming that it is referable to the mitral orifice. A murmur due to existing endocarditis is soft, usually not intense, and, as a rule, limited to a circumscribed space. Roughness and great intensity denote valvular lesions. Diffusion of the murmur over the left lateral and posterior surfaces of the chest, indicates lesions which permit regurgitation. Diastolic murmurs are usually, if not always, due to valvular lesions; consequently, a systolic murmur cannot be considered as a sign of endocarditis, if a diastolic murmur be also present. The previous occurrence of rheumatism is to be taken into account. Other things being equal, the chances that a murmur proceeds from endocardial inflammation are more if rheumatism have not occurred previously. Attention to these points will enable the practitioner to decide, not always with positiveness, but with an approximation towards certainty, whether a murmur be, or be not, significant of endocarditis.

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[1] *Vide* page 229.

A murmur developed by endocarditis generally continues not only during the continuance of the disease, but ever afterward. There are exceptions to this rule. I have known repeatedly a mitral murmur to disappear entirely after recovery from rheumatic endocarditis, when, during the progress of the disease, and for some time afterward, it had been well marked and constant. This is to be accounted for by supposing that the swelling of the valves diminishes, the lymph, fibrin, and vegetations are gradually disintegrated and washed away, and the endocardial surface is rendered smooth by the currents of blood, so that the physical conditions for the production of murmur are no longer present. But in the majority of cases the murmur not only persists, but increases rather than diminishes in intensity in proportion as valvular lesions become more and more declared. It may continue, however, for many years without any notable alteration.

What are the physical conditions incident to endocarditis which give rise to a murmur? It is probably due to roughness of the endocardial membrane covering the valves, produced by the presence of lymph, fibrin, and vegetations. It has been conjectured that, in consequence of spasmodic action of the papillary muscles, the mitral valve fails to fulfil its function, and regurgitation takes place. This is inconsistent with the constancy of the murmur and its persistence after recovery from the endocarditis. It is not necessary to assume the occurrence of regurgitation in order to account for a mitral systolic murmur. The murmur is produced in the ventricle, in other words, it is intra-ventricular, although emanating from the mitral orifice. The presence of the solid products of inflammation is sufficient to explain the occurrence of the soft, feeble, and circumscribed murmur which characterizes endocarditis.[1]

The heart-sounds may present certain abnormal modifications in endocarditis. Reduplications are sometimes observed. One or both of the sounds, the first sound more especially, may be less distinct than in health. The first sound may be wanting. Richardson, in his experiments, found that the first sound frequently disappeared for some time before a murmur was developed. It is not difficult to conceive of this in view of the great swelling of the auriculo-ventricular valves which he observed when the animals were killed during the early stage of the inflammation—the segments resembling an injected uvula, and lying so close to each other that, when the heart was contracting, they must have cushioned against each other, fulfilling their office of preventing regurgitation without tension or movement. Theoretically, it would be expected that the mitral valvular element of the first sound should be lessened or extinguished, the tricuspid valvular element remaining unimpaired. It is not improbable that this change may precede the development of a murmur, and thus be of value as an earlier physical sign than the latter.

DIAGNOSIS OF ENDOCARDITIS.

The diagnosis of endocarditis rests on auscultatory evidence. It is impossible to determine the existence of the disease by means of the symptoms alone; it is therefore necessarily overlooked by those who do not employ auscultation. The evidence consists in the development of an endocardial murmur, in connection with symptoms which corroborate its significance. In a certain proportion of cases, the diagnosis may be made with positiveness. When the development of a murmur is a matter of observation, under circumstances which render the occurrence of the disease probable, there is no room for doubt. The diagnosis is less easy, often difficult, and sometimes impossible, in cases in which the newness of a murmur is to be determined, not by observation, but inferentially. When this is the case, the practitioner is liable to err, on the one hand, in basing his diagnosis on the presence of a murmur which is not newly developed, and, on the other hand, in attributing a newly-developed murmur to other conditions than an existing inflammation.

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[1] I should add that I have observed a mitral systolic murmur to disappear before the termination of rheumatism. I have even noted the existence of this murmur during one day only, careful auscultation failing to discover any the day previous and subsequently. But in this case it seems to me more reasonable to attribute the production of the murmur to the deposit of fibrin or lymph which was soon washed away by the currents of blood, than to spasmodic action of the papillary muscles.—*Note in first edition.*

In a patient who presents unequivocal evidence of valvular lesions, the diagnosis of endocarditis is difficult, and often impossible. How is the practitioner to determine that murmurs, under these circumstances, are due to existing inflammation, and not to the valvular lesions? It is possible, if a case have been under observation previously, that certain changes in the situation and character of the murmur may be fairly attributable to superinduced endocarditis, but this will happen in only a small proportion of instances. I have met with cases repeatedly in which murmurs connected with old valvular lesions have been considered as evidence of inflammation, and a course of treatment pursued which was highly prejudicial to the welfare of the patients. But the error, in these instances, proceeded from a very imperfect knowledge of the diseases of the heart. A question as to the existence of endocarditis in connection with long-standing organic disease, can hardly arise except during an attack of acute rheumatism. Not infrequently, under these circumstances, the question does arise. A patient has had one or more previous attacks of rheumatism, which have led to organic disease of the heart. A fresh attack occurs. The symptoms and signs referable to the heart may be due wholly to the pre-existing organic disease, or they may, in part, proceed from new physical conditions incident to an existing endocardial inflammation. To determine positively with respect to this point, is certainly one of the most difficult problems in diagnosis. The problem, in fact, cannot be solved with positiveness. Cases of organic disease of heart, therefore, are to be excluded from the category of those in which a diagnosis is generally practicable.

Idiopathic endocarditis is certainly one of the rarest of diseases. But, as already stated, there is reason to believe that it occurs when it is not recognized. It would, perhaps, be discovered in some of the cases in which it is overlooked, if practitioners, in the first place, were more generally qualified to employ physical exploration, and, in the second place, if it were more the custom to auscultate the heart even when the symptoms do not point distinctly to disease of that organ. Is the diagnosis of idiopathic endocarditis practicable? An endocardial murmur developed under the observation of the practitioner, preceded and accompanied by pain or uneasiness in the præcordial region, febrile movement and excited action of the heart, other affections which might give rise to these symptoms being excluded, would warrant a positive diagnosis. It is, however, hardly to be expected that this combination of circumstances will often be presented in practice. The symptoms will not be so well declared, and at the first examination a murmur may be found, the previous duration of which is indeterminate. I confess that I have no knowledge of idiopathic endocarditis, derived from the clinical study of the disease, and older as well as better observers have made the same confession. Stokes remarks with reference to this subject: "In truth, we rarely meet with a case of simple, idiopathic endocarditis fit to be considered as a type of the signs and symptoms of the disease. Such a case, at least, has never occurred to me."[1]

Pericarditis is so frequently associated with endocarditis, that the existence of the former renders the coexistence of the latter highly probable. But in some instances of endo-pericarditis, an endocardial murmur may be, for a time, wanting, being obscured by the friction-sound, or the heart being too much weakened by the compression of liquid effusion to produce it. On the other hand, the pressure of liquid effusion and lymph on the large vessels within the pericardium has been supposed to give rise in some cases to murmur at the arterial orifices. Of course, when pericarditis exists, symptoms referable to the heart are of no value as respects the diagnosis of endocarditis. The combination of inflammation of the lining and investing membrane of the heart is more serious than either affection singly; but happily, the diagnosis of endocarditis under these circumstances does not influence materially the treatment.

To the foregoing remarks on the diagnosis of endocarditis, which are those contained in the first edition of this work, I have to add, that the existence of the affection in cases of rheumatism, as there is reason to believe, has been often predicated on insufficient evidence. Basic cardiac murmurs, that is, an aortic direct and a pulmonic direct murmur, are common in cases of rheumatism, when they are inorganic or blood-murmurs. As a rule, in females, auscultation, with the binaural stethoscope, discloses one or both of these murmurs. An aortic direct murmur, even when it appears to have been developed after a case has come under observation, cannot be considered as alone proof of endocarditis. To constitute diagnostic evidence of this disease, the murmur must be associated with well-marked symptoms pointing to endocardial inflammation, and there must be the absence of circumstances which render it probable that the murmur is inorganic. A basic murmur may be pulmonic. I have been led to think that a pulmonic direct inorganic murmur in rheumatism is quite as likely to exist alone as an aortic direct inorganic murmur; of course, a pulmonic direct murmur existing alone, whether it be organic or inorganic, is no evidence of rheumatic endocarditis. The coexistence of an aortic direct and a pulmonic direct murmur renders it probable that both are inorganic. The various circumstances which are involved in the discrimination of inorganic murmurs have been considered in a previous chapter, to which the reader is referred.[1]

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[1] Op. cit., Am. ed., p. 118.

The error of considering a basic inorganic murmur as adequate evidence of endocarditis in rheumatism, has, as I believe, vitiated, to a greater or less extent, statistics relating to the proportion of rheumatic cases in which endocarditis occurs. The proportion of nearly one-half is certainly too large, and that of Bamberger, namely, twenty per cent., is nearer the truth.[2] Another source of error is, perhaps, not very infrequent, namely, considering the fact of the presence of a mitral murmur sufficient for the diagnosis, without determining whether or not the murmur was developed in connection with the attack of rheumatism, that is, without being sure that the murmur did not exist previously.

PROGNOSIS IN CASES OF ENDOCARDITIS.

What is to be said under this head has been anticipated in the foregoing remarks. The prognosis, as regards immediate danger to life, and even the continuance of symptoms which occasion inconvenience, is favorable. It is possible that the inflammation may become chronic and persist for an indefinite period. This may be suspected, if the patient complain of uneasiness in the præcordia, and the action of the heart continue unduly excited. But our knowledge of chronic endocarditis is not sufficient to furnish grounds for discriminating between it and valvular lesions resulting from changes which take place after inflammatory action has ceased. Hence, there is no advantage in treating of acute and chronic endocarditis separately. Certain contingent or accidental events, of a serious nature, to which reference has been already made, are liable to occur during the progress of endocarditis. These are, the formation of fibrinous coagula; the detachment of vegetations or of masses of fibrin or lymph constituting emboli; the admixture of disintegrated solid deposits, and purulent infection of the blood. Clinical observation, however, shows that in a large majority of cases of endocarditis, recovery takes place without serious accidents; the symptoms referable to the heart, if any were present, disappear, leaving the patient exposed to the evils arising from valvular lesions, which may become developed at a period more or less remote.

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[1] *Vide* page 226.

[2] *Vide* page 431.

TREATMENT OF ENDOCARDITIS.

The objects of treatment in pericarditis and endocarditis are not in all respects similar. In pericarditis, the compression of the heart by the accumulation of liquid within the pericardial sac is a source of distress and danger. To prevent this accumulation, and promote its removal, are important therapeutical ends. In endocarditis, the action of the heart is free from all mechanical restraint. In pericarditis, the inflammation is more generally diffused, and a greater effect is produced upon the muscular walls, first by excitation, and afterwards by paralysis. In endocarditis, the inflammation is seated especially in the membrane connected with the valves and orifices, where it is not in contact with the muscular walls, and the latter are consequently affected in a less degree. In pericarditis, the aim of the practitioner is often to avert impending death. In endocarditis there is little fear of a fatal result. But although the two affections are so dissimilar in many respects, the general principles of management are in a great measure alike applicable to both.

The therapeutical indications in the treatment of endocarditis relate mainly to the alterations to which the membrane is exposed, and to the products of inflammation. The objects are to lessen, as far as possible, the local effects of the inflammation, to aid in restoration from these effects, and thus protect the organ from the remote consequences arising from incurable and progressive unsoundness. These objects embrace measures having in view abatement of the intensity of the inflammation, abridging its duration, limiting the exudation of lymph and the precipitation of fibrin, and effecting the removal of solid deposits. The measures for these ends are those which were involved in the treatment of pericarditis, viz., bloodletting and other antiphlogistic measures, opium, sedatives, eliminatives, and counter-irritation.

In the employment of bloodletting, the practitioner is to be guided by the same indicating and contraindicating circumstances as in other inflammations. This remedy is indicated, and the extent to which it is to be carried is to be regulated by the apparent intensity of the inflammation, the state of the vascular system, the constitution of the patient, and its immediate effects. It is contraindicated by weakness of the circulation, feebleness of the constitution, anæmia, and when, upon trial, want of tolerance of the remedy is apparent. The indications are rarely present in cases of rheumatic pericarditis, and never when the disease is developed in connection with renal disease, the eruptive fevers, pleurisy, pneumonia, and pyæmia. The remarks with respect to this remedy in pericarditis are, in general, here applicable; but the danger incident to the injudicious employment of bloodletting is greater in pericarditis, in view of the tendency of the latter to induce weakness and paralysis of the heart. Aside from the supposed effect of bloodletting in diminishing the intensity of inflammatory action, it may be useful by lessening the labor which the heart has to perform, and preventing the accumulation of blood within its cavities. Bloodletting in endocarditis, as in other inflammations, is to be employed only during the early part of the disease. It is not called for by the disease *per se*, but by the circumstances attendant on the disease. Either general or local bloodletting may be employed, the latter when it is not desired to abstract a large quantity of blood or to withdraw it rapidly. In most instances the indications for bloodletting will be fulfilled by leeching or cupping.

Other measures entering into the antiphlogistic treatment are purgation and low diet. Purgative remedies may be employed as a means of depletion when circumstances contraindicate bloodletting. The saline purgatives are best suited for this purpose. Depletion is also effected indirectly by limiting the supply of nutriment. These measures, as well as bloodletting, can only be appropriate in the early period of the inflammation. It is, to say the least, useless to continue them after the inflammation has continued sufficiently long to produce all the immediate local effects to which it is likely to give rise. After the lapse of a few days from the date of the attack, they are not indicated more than, for example, in the second or exudation stage of pneumonia.

The pain in endocarditis is rarely sufficient to call for opiates. But it is fair to infer from the apparent usefulness of opium in inflammations affecting analogous structures, that it is a useful remedy in this disease.

The nauseant sedatives and the veratrum viride may sometimes be useful in reducing the excited action of the heart. As a cardiac sedative, aconite is to be preferred. Given in small doses, with short intervals, this is an efficient and a safe remedy for reducing the excited action of the heart. Sedatives should not be carried to the extent of weakening the heart's action; for, although there is not so much immediate danger from this effect as in pericarditis, it must be unfavorable by preventing the completeness of the ventricular contractions and favoring the accumulation of blood in the cavities of the heart.

Eliminative remedies are indicated in endocarditis on precisely the same grounds and to the same extent as in pericarditis, when the disease occurs in connection with acute rheumatism or with disease of the kidneys. The treatment in these affections which is most effective in removing from the blood the poisonous principles giving rise to local inflammations will prove most effectual in preventing both the development and the persistence of endocarditis. The remarks under this head in connection with pericarditis are equally pertinent to the present subject, and need not be repeated. Certain facts observed by Richardson in his experiments are interesting with reference to the effect of eliminative remedies. In about twelve hours after the injection of the lactic acid solution into the peritoneum, when the symptoms denoting the commencement of endocarditis became developed, if the animal was freely purged or passed a large quantity of urine, the symptoms all subsided, and renewal of the injection was necessary in order to sustain the effect.

Counter-irritants, namely, sinapisms, blisters, and stimulating liniments, are indicated in the treatment of endocarditis as in pericarditis, the only difference between the two affections as regards the application of these remedies consisting in the fact that in pericarditis the absorption of liquid effusion may be promoted by vesication, while in endocarditis this is not an object of treatment.

Regarding the treatment of endocarditis from another point of view, viz., with reference to the objects or indications which are presented during the progress of the disease, the measures which have been mentioned may be recapitulated, and some additional points relating to the management noticed.

Bearing in mind the frequent occurrence of the disease in the course of acute rheumatism, it is important to prevent its development. Clinical facts demonstrate that a positive prophylactic influence is exerted by the full alkaline treatment of rheumatism. The reader is referred to remarks relating to this topic in connection with the treatment of pericarditis. In cases, also, of renal disease, the elimination of urea is important with a view to the prevention of the affection under consideration, as well as pericarditis and other uræmic effects.

At the commencement of endocarditis, and during the early part of the disease, it is an object of treatment to diminish the intensity of the inflammation. This object is important not on account of any immediate danger to life, however intense the inflammation, but in order to limit its local effects. The means for accomplishing this object are essentially those which are regarded as useful at the onset and during the early stage of inflammation affecting analogous structures. They consist of local or general bloodletting in certain cases, saline purgatives, and low diet. In pursuing these measures, the practitioner is to be guided, not by the mere fact that endocarditis exists, but by the associated circumstances in individual cases, giving due consideration to those which may contraindicate bloodletting and other modes of depletion. These measures are not to be employed or continued when the inflammation has existed for several days, the immediate local effects having then already taken place, so far as these are dependent on the intensity of the inflammation.

The solid products incident to endocarditis having been seen to consist, in part of coagulated fibrin derived from the blood contained within the cavities of the heart, and this effect being in a measure dependent on an excess of fibrin in the blood, especially when the disease occurs during the course of acute rheumatism, it is a rational object of treatment to diminish the quantity of fibrin (hyperinosis) by therapeutical measures. Mercury and alkaline remedies have been supposed to fulfil this indication, but their efficiency cannot be considered as established.

If it be true that the presence of ammonia causes or contributes to the liquid state of fibrin in the blood, ammonia as a remedy is rationally indicated. To fulfil this indication, the carbonate of ammonia may be given in full doses after short intervals. If not useful, there is no reason to suppose that this remedy, employed freely, does harm, provided it be tolerated by the stomach. Ammonia is also rationally indicated with a view to prevent the formation of large fibrinous coagula.

Pain in some cases, and more frequently excited action of the heart, call for opium. This remedy, there is reason to believe, is useful in this, as in other inflammations, not merely as a palliative of suffering, but from a power of controlling, to some extent, the inflammatory processes. Nauseant sedatives, such as antimony and the veratrum viride, are to be employed with circumspection, so as not to weaken unduly the muscular power of the organ, and aconite, as a cardiac sedative, is to be preferred.

It is an important object of treatment to prevent the persistence of endocarditis, and counter-irritants may exert more or less effect in hastening the disappearance of the inflammation.

The indications during convalescence, and subsequently, are essentially the same as during and after recovery from other inflammations affecting important organs. Avoidance of causes which may reproduce the affection is important. The powers of the heart should not be unduly tasked by violent exercise, abuse of stimulants, or excesses of any kind. It may be doubted whether an amount of physical activity necessary to vigorous health, be unfavorable as regards the liability to organic disease. A restricted diet, habits of inactivity, and other measures calculated to enfeeble the system, are more likely to hasten than postpone the development of structural lesions. It is injudicious to lead the patient to anticipate the occurrence of remote evils which he may escape, and against which, at all events, he cannot be forearmed by being forewarned. The moral effect of looking forward to organic disease of heart may prove unfavorable to a condition of mind and body which is not only conducive to present comfort, but affords, in some degree, a protection against the danger to be apprehended.

In concluding these remarks on the treatment of endocarditis, two or three practical points remain to be noticed. In a pretty large proportion of cases the inflammation is not intense; it is evidently subacute, as represented by the symptoms. These are so far from being prominent, that the disease is habitually overlooked by those who do not resort to physical exploration, and its occurrence was unknown prior to the application of auscultation to the study of cardiac affections. In these cases the expediency of active therapeutical interference is doubtful. Bloodletting and other reducing measures are of questionable propriety, and the tendency to employ heroic remedies, or to push them too far, in view of remote evils, is to be resisted. Here, as in other forms of disease, as much injury may be done by excessive, as by insufficient treatment. Another point relates to the period when the inflammation has ceased, and, consequently, the indications for treatment having reference to inflammation are no longer present. It is not always easy to determine when this period arrives. But it is important to warn the practitioner against attaching undue importance to the continuance of an endocardial murmur. This will be likely to persist, although the inflammation does not continue, for an indefinite time, and generally ever afterwards. The persistence of the murmur is no proof of inflammation, and does not, of itself, indicate the need of therapeutical measures. The symptoms must be relied upon in determining the intensity of the inflammation during the course of the disease, and the period of its cessation. The latter is declared by the disappearance of pain or uneasiness in the præcordia, absence of febrile movement, and quietude of the heart's action. Finally, the importance of not attributing to endocarditis the symptoms which may be associated with an endocardial murmur in cases of organic disease is to be enforced. I have met repeatedly with instances of valvular lesions of long standing, in which bloodletting, low diet, mercurialization, &c., had been employed with a view to combat existing inflammation. It is important to avoid this error, since, in a large proportion of the cases of organic disease of heart, these therapeutical measures are injurious.

MYOCARDITIS.

Inflammation of the muscular structure of the heart constitutes the affection called carditis or myocarditis. Treating of this affection so far as it is of interest and importance to the physician, in a practical point of view, a brief consideration will suffice, without any formal subdivision of the subject.

The muscular substance of the heart is the seat of inflammation much less frequently than the investing and lining membrane of the organ. As occurring independently of pericarditis and endocarditis, myocarditis is extremely rare. Either the pericardium or the endocardium, or both membranes, are implicated in the great majority of the cases in which the muscular tissue is found after death to present the evidences of inflammation. The inflammation, probably, in most instances, extends from the investing or lining membrane to the muscular substance; but the latter may be primarily affected. The inflammation is usually limited to certain portions of the heart, and it occurs much oftener in the left than in the right ventricle. It may be confined to the outer or inner layers of muscular fibres, or it may extend throughout the walls, and affect the *columnæ carneæ*. The septum is less liable to be affected than the ventricular walls.

If suppuration takes place, pus is found either in small collections, forming abscesses, or infiltrated more or less throughout the muscular walls. When abscesses exist, the surrounding parts present, at the same time, purulent infiltration. The formation of abscesses involves destruction of the muscular structure to a greater or less extent. They are usually formed in the left ventricle. In a case reported by Graves, a collection of two ounces of pus was found in the walls of this ventricle. The muscular substance in the parts infiltrated is livid, softened, and more or less disintegrated. Abscesses may discharge their contents into the pericardial sac, giving rise to acute pericarditis, if the latter be not already present. Or they may evacuate into the ventricular cavity, in this case giving rise to purulent infection of the blood. In either case a fatal result is inevitable. An abscess formed in the ventricular septum may lead to communication between the two ventricles. It has been conjectured that abscesses in the muscular substance of the heart may be caused by emboli passing into the coronary arteries, and that the emboli may be derived from the lung in cases of pulmonary gangrene.

Another termination of myocarditis is induration of the walls of the heart from the presence of lymph and the formation of fibroid tissue. This termination involves weakness and atrophy of the muscular substance.

An ulterior result of myocarditis is aneurismal dilatation of the walls of the heart. These have been considered in another chapter.[1] Rupture of the heart is an event in some instances incidental to inflammation of the cardiac substance.

Clinically considered, myocarditis is almost invariably associated with pericarditis, endocarditis, or endo-pericarditis, and its existence is not determinable during life. It may sometimes be suspected when the gravity of the cardiac symptoms is out of proportion to the apparent amount of endocardial or pericardial inflammation. But this statement is indefinite. There are no symptoms nor signs which warrant a diagnosis even approximating to positiveness. This remark will apply also to the very rare instances in which inflammation is limited to the muscular substance, the lining and investing membranes remaining unaffected. There would, therefore, be no advantage, practically, in dwelling on the subject. It is obvious that in proportion as myocarditis is added to endocarditis and pericarditis, singly or conjoined, the symptoms referable to the heart will denote increased gravity of cardiac disease, and the immediate danger is augmented. The patient is also exposed to certain accidents which have been mentioned, namely, rupture, aneurismal dilatation, perforation of the interventricular septum, and purulent infection of the blood. The discovery of these, during life, does not come within the reach of diagnosis.

As regards the treatment both of myocarditis and its accidents, the therapeutical measures which are likely to prove of any avail are perhaps indicated by the symptoms as clearly as if the diagnosis were practicable.

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[1] *Vide* page 115.

CHAPTER IX.

FUNCTIONAL DISORDER OF THE HEART.

Definition, and the different forms of disorder—Pathological relations and causation of functional disorder—Association with plethora, anæmia, various derangements of the nervous system, dyspepsia, gout, &c.—Symptoms of functional disorder—Physical signs furnished by percussion, palpation, and auscultation—Diagnosis of functional disorder—Prognosis—Treatment.

By functional disorder of the heart is meant disturbed action occurring independently of either inflammatory or organic affections. These affections usually involve more or less functional disorder, but the latter often occurs when the former are not present, being purely dynamic, or pertaining exclusively to the vital properties of the organ. In most instances the disturbed action of the heart is evidently due to morbid conditions seated elsewhere. It is usually symptomatic of either blood-changes, or affections of the nervous system, and, not infrequently, of both conjoined. These morbid conditions, although they are independent of inflammation and structural lesions, may, nevertheless, be associated with the latter. It is a fact important to be borne in mind, that disordered function of the heart, in certain cases of inflammatory, and, more especially, organic affections, involves the same morbid conditions which often exist independently of these affections. This is a practical point which will be again referred to. The subject of functional disorder of the heart is of great importance in a practical point of view, on account of the frequency of its occurrence, the anxiety which it occasions, and the liability of confounding it with organic disease. Of the persons who make complaint of symptoms referable to the heart, a large majority suffer from functional disorder only. But the discrimination of functional from organic affections can only be made by one who is thoroughly acquainted with the subject. The immense importance of discriminating correctly is obvious, when it is considered that structural lesions involve more or less danger, while disorder of function, although often in a high degree distressing, very rarely, if ever, proves serious.

Exclusive of the affections just named, functional disorder of the heart is not always identical, but presents certain varieties in different cases. In the mildest form of disorder, the action of the heart is simply increased unduly by transient exciting causes, such as mental emotions, muscular exercise, ingestion of food or stimulants, &c. The organ is morbidly excitable, but its action is not disturbed to an extent to occasion great inconvenience or annoyance.

Persisting inordinate action is another form of disorder. I have met with several instances in which the heart acted with regular rhythm, but with abnormal rapidity and force, irrespective of any exciting causes, the excited action continuing constantly for days, weeks, and even months. The pulse in these instances was uniformly frequent—from 110 to 120 per minute. The patients were conscious of an undue force of impulse and intensity of the heart-sounds; it was difficult for them to withdraw their attention from the action of the heart, and to overcome a conviction of the existence of organic disease. This is not a frequent form of disorder. It is observed in females much oftener than in males, and this form of functional disorder is sometimes associated with enlargement of the thyroid body, and protuberance of the eyeballs.[1]

Certain affections which have been already considered exemplify different forms of functional disorder of the heart. These affections are, palpitation with exophthalmic goitre, sometimes called Graves's disease, reduplication of the sounds of the heart, and angina pectoris. In the arrangement of topics, it was most convenient to treat of these affections directly after having treated of organic diseases of the heart, although, were strict pathological propriety considered, they would properly be embraced in this chapter.

As commonly presented in practice, functional disorder occurs in paroxysms, and the rhythm of the heart's action is disturbed. Either with or without an obvious exciting cause, the patient is conscious of a violent beating of the heart. The movements of the organ, in severe cases, are tumultuous and extremely irregular; the systolic contractions at one instant following in rapid succession, at another instant more slowly, and intermittency occurring more or less frequently. The patient is painfully conscious, not only of the morbid intensity of the action, but of the rhythmical disturbance. Absolute repose is necessary. A feeling of impending death is experienced. Great anxiety and apprehension usually accompany the paroxysms, especially at first. The terror of the patient, in fact, not infrequently enhances considerably the disorder. The paroxysms may continue for a few moments only, or for several hours. Their severity varies much in different cases. In mild cases, as when they occur in connection with hysteria, the sense of disturbance consists in fluttering movements referred to the præcordia. As regards the recurrence of the paroxysms, cases vary greatly. They may recur at short intervals, being easily provoked by various exciting causes, and occurring spontaneously; or they may take place at periods more or less remote. The disturbance of rhythm in these paroxysms is sometimes so great that, to quote the language of personification used by Bouillaud, the heart seems to be affected with a species of *insanity* (*une folie véritable*), beating at random, instead of with that regular, definite purpose which seems almost to involve a motive in its healthy action.

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[1] *Vide* page 310.

Another species of paroxysm is characterized by irregularity and intermissions, without increased force of the heart's action, but, on the contrary, the action of the heart may be quite feeble. I have observed these paroxysms to occur in a person liable to functional disorder of the heart, especially on exposure to cold, during fatigue from muscular exertion, and when the habitual time of taking food was delayed. This variety is even more distressing than that in which the paroxysms are characterized by violence of the heart's action. The feeling of impending death is rendered more vivid by a tendency to syncope.

Another paroxysmal variety consists in a sudden momentary disturbance, which is either an intermission or apparently a trembling movement of the heart, occurring at rare intervals, or more or less frequently. These paroxysms, until the mind becomes accustomed to them, inspire great terror. The patient feels, after they have passed, as if he had just escaped sudden death, and this feeling often causes the heart to beat rapidly after the paroxysms have ceased. After a time, patients become habituated to their occurrence, and they occasion much less apprehension. I have met with a great number of persons who have been subject to them for many years.

Intermittency of the heart's action, as a variety of functional disorder, is to be discriminated from intermittency existing as a congenital or an acquired peculiarity of the heart's action. In some persons the occurrence of intermissions, at either regular or irregular intervals, is natural; and they sometimes become habitual after middle age without denoting disease, and the person is not conscious of their occurrence. False intermissions are also to be distinguished from true intermittency. Intermissions are said to be false when, at either regular or irregular intervals, the force of the ventricular contraction is not sufficient to produce a radial pulsation. The pulse intermits, but there is no actual intermission of the heart's action. If auscultation be practised, the first or systolic sound is found to be comparatively weak when the pulse intermits. In a preceding chapter I have introduced two cases in which a false intermission occurred in regular alternation with a ventricular systole represented by the radial pulse.

The foregoing are the varied forms under which functional disorder has presented itself in my own clinical experience. Different varieties, however, are frequently associated in the same case. All the forms are commonly embraced under the name palpitation. They are also called, in distinction from inflammation and structural lesions, inorganic affections of the heart. As before remarked, functional disorder, in general, depends on morbid conditions seated elsewhere than in the heart. These causative conditions are by no means the same in all cases. A correct appreciation of the pathological relations of the disturbed cardiac action in individual cases is essential with reference to appropriate treatment. To these relations attention will now be directed.

PATHOLOGICAL RELATIONS AND CAUSATION OF FUNCTIONAL DISORDER OF THE HEART.

Of the different morbid conditions on which functional disorder of the heart is dependent, clinical observation shows the most important to be plethora, anæmia, derangement of the nervous system induced by various causes, dyspepsia, and the gouty diathesis.

In the condition known as plethora, in which the blood is abnormally rich in red globules, and perhaps, in excess as regards quantity, the heart appears to be overtasked and over-stimulated, and becomes, in consequence, morbidly irritable. Functional disorder, thus induced, is characterized by violence of action, without much, if any, disturbance of rhythm. Palpitation may be the first symptom of the plethoric condition, which awakens the anxiety of the patient respecting the state of his health. His attention is usually at once concentrated on the heart, and he is fearful of organic disease. Cases which fall under this head are presented in persons who have altered their mode of life, exchanging habits of physical activity for sedentary pursuits or luxurious leisure. Students coming from the farm or workshop, men of business retiring to live in ease, and all who, in addition to indolence, cultivate the pleasures of the table, are liable, among other evils, to suffer from functional disorder of the heart incident to plethora. These cases are to be discriminated, with reference to the proper treatment, from others in which the pathological relations are quite different.

Cases of functional disorder are much oftener met with in connection with a condition the opposite of plethora, namely, anæmia. It is rare for well-marked anæmia to exist without more or less disturbance of the heart's action. Cases belonging to this class occur vastly oftener among females than males, anæmia being as infrequent with the latter, as it is common with the former. Anæmia being produced by hemorrhages, leucorrhœa, frequent childbearing, prolonged lactation, &c., the functional disorder of the heart will, of course, in individual cases, be referable to one or more of these ulterior pathological relations, the anæmia, however, being the intervening causative condition. But the degree of disorder is not always proportionate to the anæmia, being sometimes slight when the anæmic state is marked, and, conversely, severe in some cases in which the latter is scarcely appreciable. Anæmia giving rise to a multitude of morbid effects, in addition to disturbance of the heart's action, more or less of these are associated with the latter. Patients with functional disorder dependent on anæmia will be likely to present as symptoms, either coexisting or developed in succession, coldness of the extremities, cephalalgia, intercostal neuralgia, and neuralgic affections in different situations, depression of spirits, &c. Of all the associated morbid effects of anæmia, the cardiac disorder often occasions the most annoyance and anxiety. The fear of organic disease and of sudden death is added to the distress which belongs intrinsically to the disorder. In cases of marked anæmia, patients are frequently supposed to labor under organic disease of the heart by those who trust exclusively to symptomatic phenomena in diagnosis. The symptoms, in fact, sometimes point strongly to the existence of organic disease. Not infrequently, palpitation is excited by the slightest exertion; dyspnœa is experienced; pain or uneasiness is referred to the præcordia; the countenance is morbid, and, occasionally, the hydræmic condition of the blood leads to œdema and anasarca. I have met with several instances in which all the symptoms of advanced organic disease of the heart were simulated by the morbid effects of anæmia induced by prolonged lactation and other causes. The importance of a correct diagnosis, as regards the prognosis and treatment, in these cases, is truly immense.

Derangement of the nervous system is doubtless the immediate cause of cardiac disorder in cases of anæmia. The morbid condition of the blood leads to disturbance of the heart's action through the intervention of the nervous system. But the latter may be deranged and functional disorder of the heart produced by various causes, irrespective of anæmia. Cases of hysterical palpitation come under this head. Hysteria is frequently, but by no means invariably, associated with anæmia. It occurs in the plethoric. Functional disorder of the heart is one of the commonest of the varied phenomena included under the name hysteria. Disturbed action of the heart is often a prominent feature of the hysterical condition. Various morbid agencies induce a state of nervous derangement, of which functional disorder of the heart is a distressing manifestation. Venereal excesses and the solitary vice are frequent causes. In the endeavor to trace this and other effects of derangement of the nervous system to their source, the practitioner should not omit inquiries as to sexual indulgence in the married, for there are persons who appear to think that any amount of legitimate indulgence is physically innocent; and when questioned, will confess to having practised one or more acts of coition daily for a series of years.

The excessive use of tobacco is another cause of nervous derangement giving rise to functional disorder of the heart. This is a frequent cause. Many persons are led by their experience to observe that after an unusual indulgence in this luxury, they are apt to suffer from palpitation, and the disorder is sometimes removed by simply discontinuing this indulgence. Strong tea in some persons occasions severe paroxysms of palpitation. Stokes has cited several striking illustrations.[1] Strong coffee induces this effect in certain conditions of the system, or in consequence of a peculiarity of constitution. Excessive mental exercise and protracted vigilance belong in this category; and, more than all, long-continued anxiety or distress of mind. In a pretty large proportion of the cases of functional disorder of the heart, it is traceable to nervous derangement induced by mental causes. Persons are especially prone to this disorder who are so constituted that, whatever may be the circumstances surrounding them, they are constantly anxious and worried. In persons not thus unhappily constituted, the disorder may originate in the severe afflictions, calamities, and disappointments to which human life is exposed. Whatever may be the causes inducing that derangement of the nervous system which leads to disturbed action of the heart, mental depression is generally a prominent symptom. The conviction of the existence of organic disease is often with great difficulty removed. The patient sometimes persists in this conviction in spite of the strongest assurances of the physician. His attention is occupied much of the time in watching the action of the heart. He acquires the habit of feeling the pulse or the beating in the præcordial region. He lives in daily apprehension of sudden death. This truly pitiable condition tends, in no small degree, to aggravate the nervous derangement, and thus reacts on the cardiac disorder. Every one who has been brought much into contact with students of medicine, must have been led to remark the frequency with which they imagine themselves to be affected with disease of the heart. The study of the diseases of this organ tends to direct attention to the subject and excite their fears, if, from any cause or combination of causes, functional disorder is produced; and the dread of these diseases seems, in some instances, to be alone sufficient to induce disturbed action of the organ. A fixed belief that the heart is diseased is one of the commonest of the delusions incident to melancholia and hypochondriasis.

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[1] Stokes devotes a section to "Disturbance of the heart caused by the use of tea." *Vide* On Diseases of the Heart and Aorta, Am. ed., p. 533.

Disorder of the heart often accompanies dyspeptic ailments. Paroxysms of palpitation are frequently referable to a fit of indigestion. The latter may be the immediate determining cause in cases in which the disorder involves other causative conditions than dyspepsia. Dyspeptic ailments, in fact, in a large proportion of cases, proceed from derangement of the nervous system, induced especially by mental distress or anxiety; and it is not easy to say, under these circumstances, to what extent the cardiac disorder is dependent on a morbid condition of the stomach. Dyspeptics who suffer from disturbed action of the heart are apt to insist perseveringly on the existence of organic disease, and to cherish the most gloomy forebodings. They fall into the baneful habit of watching the action of the heart by placing the hand over the præcordia or on the pulse, and listening, at night, to the cardiac sounds. Under these circumstances they find evidence of disorder, because the anxious expectation of finding it is often sufficient to produce it.

The accumulation of gas in the stomach, when other dyspeptic symptoms are not present, seems often to produce or increase cardiac disorder. This may be owing to mechanical pressure upon the heart. Patients suffering under paroxysms of palpitation frequently make voluntary efforts to expel wind from the stomach by belching, and express relief when they succeed in these efforts. Carminative remedies, in many instances, are useful in this way. Gastric distension, in many cases of hysteria, aggravates the symptoms referable to the heart.

The gouty diathesis involves a liability to functional disorder of the heart. Paroxysms are apt to precede other manifestations of this diathesis, occurring before any affection of the joints takes place, and perhaps ceasing to recur after the latter becomes established. Palpitation is sometimes a premonition of an approaching fit of gout. It may occur also in the intervals between the gouty attacks. The disorder, in persons subject to gout, may be due to other morbid conditions—for example, plethora; but it is reasonable to conclude, from the relations often observed to exist between the disturbed action of the heart and the arthritic attacks, that the former arises from the accumulation in the blood of the poisonous agent—supposed to be lithic acid—which gives rise to the latter. This view of the pathology has an obvious practical bearing on the management.

Other pathological relations of functional disorder of the heart have been noticed by clinical observers. It occurs during convalescence from fevers. Persons affected with deformities of the chest seem to be more liable to it. Corrigan and Forget have noticed its frequent occurrence in young persons when growth is unusually rapid. It is, apparently, sometimes induced by excessive muscular exercise. An abnormally small size of the heart has been supposed to contribute to its production. It is probable, however, that, when developed under these and other circumstances which might be added, the immediate causative conditions are included under the several classes which have been noticed, consisting of abnormal changes pertaining to the blood, or derangement of the nervous system induced by various morbid agencies, or disturbing influences, transmitted, by sympathy, from other organs.

Dr. Henry Hartshorne has described a form of functional disorder, observed by him among soldiers in the late civil war in this country, which he calls "muscular exhaustion of the heart." This form of disorder was characterized by rapidity and fulness of the heart's action while the patient was at rest, and great acceleration on the slightest exertion. It was not attributable to anæmia, the use of tobacco, or any of the usual causes of functional disorder, but, in the opinion of Dr. Hartshorne, to long-continued over-exertion, with deficiency of rest, and often of nourishment. To these, perhaps, should be added, the mental excitement, anxiety, and inquietude, which must be incident to very active service in the field when soldiers have recently entered the army from civil life. The functional disorder in these cases was long protracted. After several months of rest and treatment in hospitals, patients improved, but did not recover sufficiently to return to active field duty. A similar form of functional disorder, occurring among soldiers during the war, has been described by Prof. Alfred Stillé.[1] Hartshorne states that this affection was commonly known, among hospital surgeons, as the "trotting heart."

There is a marked difference among different persons in health as regards the excitability of the heart. This is shown by the fact that there is a wide difference in the degree of transient palpitation produced in different healthy persons by the same amount of emotional excitement, physical exercise, or ingested stimulants. Some persons have naturally so much excitability of this organ, that they may be said to have "irritable hearts;" and this peculiarity renders them more prone to the production and the persistence of functional disorder.

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[1] *Vide* Address before the Philadelphia County Medical Society, delivered February 11th, 1863.

An interesting inquiry, arising in connection with the causation and pathological relations of functional disorder of the heart, relates to the different nerves through which the morbific agencies may exert their effects? The innervation of the heart is derived, in part, from the ganglionary or sympathetic system, and partly from the pneumogastric nerves. Physiological researches seem to show that for its active force the heart is dependent on the cardiac ganglions and the plexus of nerves with which they are connected, the question whether the innervation maintaining this active force originates in the ganglions or is due to a venous influx from without the heart, being *sub judice.* That the function of the pneumogastrics, as regards the heart, is to regulate its action, appears to be established. The latter presides especially over the rhythm of the heart's movements. It is probable that morbific agencies giving rise to functional disorder may affect separately these two sources of the innervation of the heart. Causes which simply increase the frequency and force of the heart's action, with little or no disturbance of rhythm, for example, plethora, it may be supposed, act through the sympathetic system; on the other hand, causes which occasion especially disturbance of rhythm, it may be supposed, act through the pneumogastrics. The irregularity, feebleness, and intermittency which characterize the disorder in certain cases are analogous to the effects of dividing the pneumogastrics in experiments upon animals. Certain morbific agencies, namely, anæmia, toxæmia, and mental depression, probably produce their effects chiefly through the pneumogastrics. Not infrequently, however, the characters of the functional disorder show morbific influences through both sources of innervation; that is, the force of the heart's action is increased, and the rhythm of its movements is also disturbed. Most of the causes of functional disorder probably act primarily upon the nervous centres; but in some cases, for example, when the disorder depends on dyspepsia, a morbid influence is transmitted to the nervous centre, and reflected upon the heart.

SYMPTOMS ASSOCIATED WITH FUNCTIONAL DISORDER OF THE HEART.

The symptomatic phenomena in cases of functional disorder, aside from those referable directly to the heart, differ according to its various pathological relations and causes. Associated with plethora, it is accompanied by symptoms denoting vascular fulness, such as a strong pulse, a flushed face, cephalalgia from determination of blood to the brain, &c. In connection with anæmia, the attendant phenomena indicate feebleness of the circulation; the lips are pallid, the pulse small and quick, the extremities cold, &c. Dependent on certain derangements of the nervous system, it forms, in some cases, one of the multifarious elements of hysteria; in other cases, hypochondriasis, melancholia, and other symptoms referable to this system, are prominent. As incidental to dyspepsia, it is conjoined with notable disorder of the digestive functions. Occurring in persons subject to gout, it is either combined or it alternates with the varied ailments incident to this diathesis. These diversified phenomena are not properly symptoms of the cardiac disorder, but pertain to the different morbid conditions which give rise to it. And, in fact, as already stated, functional disorder of the heart is merely a symptom of these morbid conditions, and not entitled strictly to be considered as a cardiac affection. There are certain points, however, pertaining to symptomatology, which are of importance in discriminating functional disorder from organic disease. These it will be most convenient to notice in connection with the subject of diagnosis.

PHYSICAL SIGNS OF FUNCTIONAL DISORDER OF THE HEART.

Physical exploration will be seen more fully under the head of diagnosis to be of immense value in cases of functional disorder of the heart, as showing the absence of the signs of inflammatory and organic affections. The information which it affords is not less positive than if there were certain signs characteristic of functional disorder. The results of physical explorasuccession, coldness of the extremities, cephalalgia, intercostal neuralgia, and neuralgic affections in different situations, depression of spirits, &c. Of all the associated morbid effects of anæmia, the cardiac disorder often occasions the most annoyance and anxiety. The fear of organic disease and of sudden death is added to the distress which belongs intrinsically to the disorder. In cases of marked anæmia, patients are frequently supposed to labor under organic disease of the heart by those who trust exclusively to symptomatic phenomena in diagnosis. The symptoms, in fact, sometimes point strongly to the existence of organic disease. Not infrequently, palpitation is excited by the slightest exertion; dyspnœa is experienced; pain or uneasiness is referred to the præcordia; the countenance is morbid, and, occasionally, the hydræmic condition of the blood leads to œdema and anasarca. I have met with several instances in which all the symptoms of advanced organic disease of the heart were simulated by the morbid effects of anæmia induced by prolonged lactation and other causes. The importance of a correct diagnosis, as regards the prognosis and treatment, in these cases, is truly immense.

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The gouty diathesis involves a liability to functional disorder of the heart. Paroxysms are apt to precede other manifestations of this diathesis, occurring before any affection of the joints takes place, and perhaps ceasing to recur after the latter becomes established. Palpitation is sometimes a premonition of an approaching fit of gout. It may occur also in the intervals between the gouty attacks. The disorder, in persons subject to gout, may be due to other morbid conditions—for example, plethora; but it is reasonable to conclude, from the relations often observed to exist between the disturbed action of the heart and the arthritic attacks, that the former arises from the accumulation in the blood of the poisonous agent—supposed to be lithic acid—which gives rise to the latter. This view of the pathology has an obvious practical bearing on the management.

Other pathological relations of functional disorder of the heart have been noticed by clinical observers. It occurs during convalescence from fevers. Persons affected with deformities of the chest seem to be more liable to it. Corrigan and Forget have noticed its frequent occurrence in young persons when growth is unusually rapid. It is, apparently, sometimes induced by excessive muscular exercise. An abnormally small size of the heart has been supposed to contribute to its production. It is probable, however, that, when developed under these and other circumstances which might be added, the immediate causative conditions are included under the several classes which have been noticed, consisting of abnormal changes pertaining to the blood, or derangement of the nervous system induced by various morbid agencies, or disturbing influences, transmitted, by sympathy, from other organs.

Dr. Henry Hartshorne has described a form of functional disorder, observed by him among soldiers in the late civil war in this country, which he calls "muscular exhaustion of the heart." This form of disorder was characterized by rapidity and fulness of the heart's action while the patient was at rest, and great acceleration on the slightest exertion. It was not attributable to anæmia, the use of tobacco, or any of the usual causes of functional disorder, but, in the opinion of Dr. Hartshorne, to long-continued over-exertion, with deficiency of rest, and often of nourishment. To these, perhaps, should be added, the mental excitement, anxiety, and inquietude, which must be incident to very active service in the field when soldiers have recently entered the army from civil life. The functional disorder in these cases was long protracted. After several months of rest and treatment in hospitals, patients improved, but did not recover sufficiently to return to active field duty. A similar form of functional disorder, occurring among soldiers during the war, has been described by Prof. Alfred Stillé.[1] Hartshorne states that this affection was commonly known, among hospital surgeons, as the "trotting heart."

There is a marked difference among different persons in health as regards the excitability of the heart. This is shown by the fact that there is a wide difference in the degree of transient palpitation produced in different healthy persons by the same amount of emotional excitement, physical exercise, or ingested stimulants. Some persons have naturally so much excitability of this organ, that they may be said to have "irritable hearts;" and this peculiarity renders them more prone to the production and the persistence of functional disorder.

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[1] *Vide* Address before the Philadelphia County Medical Society, delivered February 11th, 1863.

An interesting inquiry, arising in connection with the causation and pathological relations of functional disorder of the heart, relates to the different nerves through which the morbific agencies may exert their effects? The innervation of the heart is derived, in part, from the ganglionary or sympathetic system, and partly from the pneumogastric nerves. Physiological researches seem to show that for its active force the heart is dependent on the cardiac ganglions and the plexus of nerves with which they are connected, the question whether the innervation maintaining this active force originates in the ganglions or is due to a venous influx from without the heart, being *sub judice*. That the function of the pneumogastrics, as regards the heart, is to regulate its action, appears to be established. The latter presides especially over the rhythm of the heart's movements. It is probable that morbific agencies giving rise to functional disorder may affect separately these two sources of the innervation of the heart. Causes which simply increase the frequency and force of the heart's action, with little or no disturbance of rhythm, for example, plethora, it may be supposed, act through the sympathetic system; on the other hand, causes which occasion especially disturbance of rhythm, it may be supposed, act through the pneumogastrics. The irregularity, feebleness, and intermittency which characterize the disorder in certain cases are analogous to the effects of dividing the pneumogastrics in experiments upon animals. Certain morbific agencies, namely, anæmia, toxæmia, and mental depression, probably produce their effects chiefly through the pneumogastrics. Not infrequently, however, the characters of the functional disorder show morbific influences through both sources of innervation; that is, the force of the heart's action is increased, and the rhythm of its movements is also disturbed. Most of the causes of functional disorder probably act primarily upon the nervous centres; but in some cases, for example, when the disorder depends on dyspepsia, a morbid influence is transmitted to the nervous centre, and reflected upon the heart.

SYMPTOMS ASSOCIATED WITH FUNCTIONAL DISORDER OF THE HEART.

The symptomatic phenomena in cases of functional disorder, aside from those referable directly to the heart, differ according to its various pathological relations and causes. Associated with plethora, it is accompanied by symptoms denoting vascular fulness, such as a strong pulse, a flushed face, cephalalgia from determination of blood to the brain, &c. In connection with anæmia, the attendant phenomena indicate feebleness of the circulation; the lips are pallid, the pulse small and quick, the extremities cold, &c. Dependent on certain derangements of the nervous system, it forms, in some cases, one of the multifarious elements of hysteria; in other cases, hypochondriasis, melancholia, and other symptoms referable to this system, are prominent. As incidental to dyspepsia, it is conjoined with notable disorder of the digestive functions. Occurring in persons subject to gout, it is either combined or it alternates with the varied ailments incident to this diathesis. These diversified phenomena are not properly symptoms of the cardiac disorder, but pertain to the different morbid conditions which give rise to it. And, in fact, as already stated, functional disorder of the heart is merely a symptom of these morbid conditions, and not entitled strictly to be considered as a cardiac affection. There are certain points, however, pertaining to symptomatology, which are of importance in discriminating functional disorder from organic disease. These it will be most convenient to notice in connection with the subject of diagnosis.

PHYSICAL SIGNS OF FUNCTIONAL DISORDER OF THE HEART.

Physical exploration will be seen more fully under the head of diagnosis to be of immense value in cases of functional disorder of the heart, as showing the absence of the signs of inflammatory and organic affections. The information which it affords is not less positive than if there were certain signs characteristic of functional disorder. The results of physical exploration are, in fact, to be considered under a twofold aspect, viz., *first*, as to the absence of abnormal phenomena which denote structural changes; and, *second*, as to the presence of the normal phenomena denoting soundness of the organ. In the latter point of view the evidence is positive, in the former it is negative. It is of use, practically, to keep this distinction in mind. In exploring the chest, the practitioner has always two objects in view. One object is to ascertain whether certain well-established signs of disease are either present or wanting; another object is to satisfy himself as to the presence of the healthy signs. To illustrate this distinction, if solidification of lung be suspected, auscultation is practised in order to discover the respiratory sign of solidification, viz., the bronchial respiration. Now, every practical auscultator knows that the lung may be solidified, and yet this sign of solidification be wanting. The evidence against solidification, therefore, is not complete when it is found that this sign is absent. But let it be ascertained that, in place of a bronchial respiration, the normal respiratory murmur continues, here is proof positive of the non-existence of solidification. This principle will be found to apply to the employment of physical exploration with a view to determine whether certain symptoms referable to the heart proceed from organic disease or from merely functional disorder.

Of the several methods of exploration, percussion, palpation, and auscultation furnish important information in cases of functional disorder.

By percussion it is ascertained that the heart is not enlarged. Functional disorder, it is true, may coexist with cardiac enlargement, the combination being due merely to coincidence. It does not follow because the heart is found to be enlarged and other lesions of structure are present, that functional disorder, irrespective of the organic disease, does not exist. But absence of enlargement is evidence that the disorder is purely functional; for clinical experience teaches that in cases of disturbed action of the heart arising from organic disease, the latter generally has induced enlargement of the organ. Percussion, therefore, is of great utility in the discrimination of functional disorder from affections involving lesions of structure.

The abnormal force of the heart's action is ascertained by palpation. The impulsion in severe paroxysms of palpitation is often violent; the whole præcordia is agitated; the organ seems to strike a forcible blow against the thoracic walls. The irregularity of the movements of the organ is also appreciated by the hand. These are merely signs of increased and disturbed *action* due to morbid excitement of the heart. They do not indicate the augmented *power* of the organ, which characterizes hypertrophy. The impulse in hypertrophy denotes strength rather than force; it is not quick and violent, but sluggish and strong; it does not give the sensation of a shock or blow, but it causes a gradual and strong heaving of the præcordia. The characters obtained by palpation, which distinguish functional excitement of the heart from enlargement by hypertrophy, are sufficiently well-marked, and have been mentioned already in treating of the latter.[1] The discrimination, however, does not rest on this distinction, for the fact that enlargement exists, in cases of hypertrophy, is determined by the coexistence of other signs. But it is to be borne in mind that functional disorder dependent on some of the morbid conditions which give rise to it independently of organic disease, may be associated with hypertrophy, and, under these circumstances, the excited action due to the former, and the increased power due to the latter, are combined.

Palpation shows, in cases of functional disorder exclusive of organic disease, that the point of apex-beat is in its normal situation; not elevated as in pericarditis with effusion, nor lowered and carried to the left as in cases of enlargement of the left ventricle.

Purring tremor, or thrill, is said to be sometimes perceived at the base of the heart in cases of purely functional disorder. This must be extremely rare. Well-marked thrill is to be considered as a sign of hypertrophy of the left ventricle combined with valvular lesions.

Auscultation furnishes important information, *first*, negatively, by showing the absence of adventitious sounds indicative of valvular lesions, and, *second*, positively, by showing that the natural sounds preserve their essential characters and normal relations to each other.

As regards adventitious sounds, the question arises, may not an endocardial murmur be produced by functional disorder alone? It is supposed that a mitral systolic murmur sometimes occurs in paroxysms of palpitation, in consequence of a spasmodic action of the papillary muscles connected with the mitral valve, interfering with the action of the latter sufficiently to permit a certain amount of regurgitation irrespective of any valvular lesions. Without denying the possibility of this occurrence, it must be extremely rare, and a murmur thus produced is necessarily either intermittent or of transient duration. A murmur referable to the mitral orifice, in the vast majority of instances, proceeds from physical changes, although these may be trivial as regards any immediate morbid effects; and if the murmur be persistent, it certainly denotes lesions, either innocuous or otherwise. At the arterial orifices, viz., the pulmonic and aortic, a murmur is often present in connection with functional disorder of the heart, when there are no valvular lesions in these situations. This murmur is therefore inorganic, and is dependent on the condition of the blood. The very frequent association of functional disorder with anæmia, accounts for the frequency of the murmur. May not the murmur in some instances be dynamic, that is, due to the excited action of the heart, without involving an abnormal condition of the blood? The affirmative is not improbable, but it is difficult to answer this inquiry positively, and practically it is not very important to do so. The question to be settled, clinically, in individual cases is, whether a murmur referable to the aortic or pulmonic orifice, coexisting with disturbed action of the heart, be organic or inorganic. The points involved in the discrimination of organic and inorganic murmurs have been considered in a preceding chapter.[1] These points may be here briefly recapitulated. An inorganic murmur is always systolic, and rarely rough in quality. Assuming that it is produced at the arterial orifices, and therefore seated at the base of the heart, it may be referred to the aorta or pulmonic artery, either or both; if the latter, this fact renders its inorganic character almost certain, provided congenital valvular lesions are excluded. An arterial murmur is heard over the carotids, and perhaps over other large arteries which are accessible. Venous hum in the veins of the neck is a constant concomitant. The murmur is usually feeble, and variable in intensity; it is often intermittent. The palpable evidences of anæmia are usually present, and it occurs much oftener in females than in males. An organic murmur, on the other hand, may be diastolic, or systolic and diastolic murmurs may be combined. It is often rough and sometimes musical. It is referable to the aortic orifice, if not to the mitral, unless it be dependent on congenital valvular lesions. If not propagated into the carotids, murmur in this, as well as in other arterial trunks, may be wanting. Venous hum may not coexist. The murmur is persistent and less fluctuating as regards intensity.

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[1] *Vide* Chapter I, p. 51.

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[1] Chapter IV, p. 226.

Attention to these differential points will generally enable the practitioner to discriminate correctly between an organic and inorganic murmur; but this discrimination, practically, with reference to the question, whether disturbed action of the heart be due purely to functional disorder, or not, is of less importance than might at first be supposed. The disturbance is probably dependent on functional disorder, whether an existing murmur be organic or inorganic, if the heart be not enlarged. It may be stated, as a rule, that valvular lesions do not give rise to notable disturbance of the heart's action prior to more or less enlargement. Hence, cardiac disorder in a marked degree, when valvular lesions exist, is attributable to abnormal conditions which are independent of the latter. A fact, already repeated more than once, is not to be lost sight of, namely, the causes of functional disorder may be superadded to organic disease; in other words, structural lesions do not render the heart exempt from the liability to become functionally disordered in consequence of the same causes which occasion disturbance of its action when it is structurally sound.

The heart-sounds in cases of functional disorder are intensified in proportion to the increased force of the heart's action. Their intensity is often such that they are perceived by the patient with great distinctness, especially at night. The valvular element of the first sound is in some cases unusually marked. There are two reasons for this. It is owing, in the first place, to the force and quickness of the ventricular contractions; and, in the second place, to the comparatively small quantity of blood within the ventricles at the time of the ventricular systole. The last-named reason obtains in a certain proportion of cases. When the ventricular contractions are rapid, there is not time between the successive contractions for the accumulation of much blood within the ventricles; the consequence is, the auriculo-ventricular valves are not floated out, the range of their movements with the systole of the ventricles is greater, and the valvular element of the first sound is intensified.[1] The predominance of the valvular element of the first sound may thus occur in opposite conditions as respects the muscular action of the heart, namely, when it is enfeebled, and when it is excited. In the latter case the predominance of this element is due to its intensity being increased out of proportion to the increase of the intensity of the element of impulsion. In the former case, the valvular element may not be actually intensified, but the element of impulsion is weakened or lost. The first sound, more than the second, is affected in its intensity by the vital condition of the heart. This sound is relatively weakened, and may be suppressed when the muscular power of the organ is greatly reduced. On the other hand, it becomes the accentuated sound at the base, and at points removed from the præcordial regions, when the muscular action is increased by morbid excitement. The integrity of the heart-sounds, the normal relative intensity of the aortic and pulmonic second sound, and of the mitral and tricuspid elements of the first sound, constitute important evidence, in cases of disturbed action of the heart, that the latter is due to simply functional disorder.

The apex-beat, or systolic sound of the heart, is sometimes accompanied by a ringing intonation called by Laennec *cliquement metallique*, or metallic tinnitus. This is occasionally observed to some extent, in health, especially in young persons, even when the heart is tranquil. It is, however, in general, a sign of excited action of the organ. It may be imitated by making light percussion on the back of the hand, the palmar surface being applied over the ear. Hope explains the production of this metallic ringing sound by supposing that "the heart in gliding forwards and upwards during its systole strikes with its apex against the *inferior margin* of the fifth rib, and thus creates an accidental sound, attended by *cliquetis* when the blow is smart." He adds: "It may be prevented at pleasure by pressing the edge of the stethoscope or anything else into the intercostal space by which that space is put, internally, on the same plane as the rib, over which the heart then glides without catching." If this be the correct explanation, inasmuch as the heart does not move upwards and forwards during its systole, the sound must be due to the apex impinging against the upper margin of the sixth, rather than the lower margin of the fifth rib, that is, assuming the point of apex-beat to be in the fifth intercostal space, as it is in the majority of persons. Whatever be the explanation, clinical observation shows that the sign occurs when the action of the heart is abnormally quick and forcible; and that it is produced by the movements of the apex against the thoracic walls, can hardly be doubted. It may occur in cases of hypertrophy, but it is more apt to be developed in connection with merely functional disorder, and it is, therefore, to some extent, significant of the latter. It was stated by Hope, that he never found it to occur in any but the meagre. It occurs certainly very seldom in persons whose chests are thickly covered with muscle or fat. Tympanitic distension of the stomach contributes to the intensity and clearness of the sound, and it may occur only under this condition. In a case observed by Walshe the sound was so loud as to be a source of alarm to the patient. Stokes remarks, and justly, that it is more common in cases in which the heart acts with great force combined with regularity of action, than in the irregularly acting hearts. As a physical sign, tinnitus is not of much practical value, since it may occur when the heart is excited, and even when it is tranquil, in health, and since it occurs in cases of enlargement as well as of merely functional disorder, although more frequently in the latter. It is perhaps important to warn the inexperienced auscultator not to attach to it a degree of significance as a morbid sound to which it is not entitled.

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[1] *Vide* page 68.

DIAGNOSIS OF FUNCTIONAL DISORDER OF THE HEART.

The diagnosis of functional disorder of the heart involves in all cases the question whether organic disease be or be not present. The symptoms referable to the heart are sufficiently explicit as to their source. The patient as well as the physician, is able at once to determine their cardiac origin. But whether they proceed merely from disturbed action, or are due to a structural affection is not so easily determined. The question is one of great practical importance. If there be only functional disorder, the physician is warranted in giving positive assurances of the absence of danger, and in holding out confident expectations of recovery. If organic disease be present, such assurances and expectations are not admissible. An intelligent patient is sufficiently aware of the difference between organic disease and functional disorder to appreciate its great importance; and he anxiously appeals to the physician for positive information with respect to this point. The ability to say positively that organic disease does not exist, often enables the physician to exert a moral influence of no mean value upon the continuance of the malady, as well as in rendering it more supportable. Errors in diagnosis are quite common. Instances have repeatedly come under my observation in which patients suffering only from disturbed action of the heart, having been told that they were affected with organic disease, have lived for months or years under a sense of danger of sudden death, a condition of mind highly conducive to the perpetuation of the disorder. On the other hand, it is not uncommon for the symptoms connected with structural lesions to be imputed to merely functional disorder. The latter error, although less unfortunate as regards its consequences than the former, sometimes leads to evil results. If the physician be not confident in his ability to decide as to the existence or non-existence of organic disease, but is sufficiently prudent not to commit himself to any conclusion, he loses the advantage which he might avail himself of, assuming the affection to be merely functional, and the patient naturally construes his reserve or indecision into an unfavorable opinion. In short, there are few problems in clinical medicine more important than that which calls for a decision as to the existence of a purely functional disorder of the heart, or an organic affection; and this problem cannot fail to present itself very frequently in medical practice. Cases of organic disease of the heart are not infrequent, and cases of merely functional disorder are exceedingly common. The importance of the diagnosis must be felt almost daily by the reflecting and conscientious practitioner.

But the diagnosis involves more than the question whether disease be or be not present. Functional disorder may be superadded to organic disease. The latter may exist, but not to an extent to occasion immediate inconvenience or danger, the symptomatic phenomena being due to disturbed action arising from morbid conditions which are independent of the structural lesions which happen to coexist. The fact that functional disorder and organic disease may be associated, and the former not dependent on the latter, is not to be lost sight of. Hence, it is not enough to decide that organic disease is present; the question then arises, Is this organic disease the source of all the symptomatic phenomena referable to the heart, or are they not due, in a greater or less degree, to functional disorder dependent on morbid conditions which have no connection with the cardiac lesions? This is a question of great importance, which is to be considered in the cases of disturbed action of the heart, in which the evidence of organic disease is found to coexist.

The objects in diagnosis, then, are, *first*, to determine whether organic disease be or be not present; and, *second*, if organic disease be present, to determine whether superadded functional disorder be not the source of more or less of the symptoms referable to the heart.

The symptoms referable to the heart, separately or combined, cannot afford positive evidence in any case that cardiac disorder is purely functional. Yet there are several points pertaining to the symptomatology, exclusive of physical signs, which are consistent with the supposition of the existence of functional disorder rather than of organic disease. These points are to be considered with reference to the diagnosis.

The mental condition is of some importance in a diagnostic point of view. Functional disorder generally occasions, in a marked degree, anxiety and apprehension. The patient is often much agitated by the idea of an examination, and awaits the result with fear and trembling. It is not infrequently difficult to convince him that he has not an organic affection, and he sometimes solicits repeated examinations lest something may have been overlooked. On the contrary, patients affected with organic disease are often, in a marked degree, apathetic on the subject. They are inclined to think that their ailments proceed from some other organ than the heart, for example, the liver or the stomach. They generally bear being told that the heart is unsound, without emotion, and frequently with apparent indifference. The contrast in the state of the mind with reference to the question as to the existence or non-existence of organic disease, is very striking.

The symptoms due to disordered action of the heart from organic disease, viz., palpitation, irregularity, intermittency, &c., occasion, as a rule, far less inconvenience than when similar symptoms arise from merely functional disorder. It is surprising, in some cases, to what extent the action of the heart is disturbed in connection with structural lesions, without the patient apparently being conscious of it. Power of impulse sufficient to raise the præcordia, and jar the whole body, is sometimes unnoticed. Irregular and intermittent action does not excite fear of sudden death. It is otherwise with cases of functional disorder. Palpitation, in these cases, causes great distress; and rhythmical disturbance produces fear that the action of the heart may be suspended, and a feeling of impending death. The positive suffering from symptoms referable to the heart, and the mental condition, furnish strong presumptive evidence of the existence of merely functional disorder.

The paroxysmal character of functional disorder, and the complete exemption, at certain periods, from cardiac disturbance, are important diagnostic points. Structural lesions, being permanent, if they be sufficient to occasion much obstruction or regurgitation, or both, induce, at length, certain symptoms which are constant, such as feebleness, irregularity and intermittency of the pulse, dyspnœa on exercise, &c. Functional disorder, on the other hand, occurs, generally, in well-marked paroxysms, and after these have ceased, the action of the heart may be natural, and there are no symptoms referable to this organ which are habitually present. A patient who is able, at any time, to take active exercise without undue excitement of the heart, or dyspnœa, may be presumed to be free from organic disease. But it is not safe to rely on the statements of patients with respect to this point, for persons affected with organic disease are often unconscious of these effects of exercise, when they are sufficiently apparent to others. Persons liable to functional disorder often are not only able to engage, without discomfort, in pursuits requiring great muscular exertion, but they are less likely, under these circumstances, to suffer from cardiac disturbance. The obvious benefit of active exercise thus becomes, in some measure, diagnostic. But the want of ability to take active exercise is by no means proof that organic disease exists, for in some cases of functional disorder associated with anæmia, slight exertion may induce palpitation, dyspnœa, &c.

Certain symptomatic events belong especially to the clinical history of organic affections, and not to that of functional disorder. Thus, general dropsy very rarely occurs in connection with the latter. This is true of lividity, hæmoptysis, paralysis from embolism, &c. These events point to the existence of organic disease, but their absence does not prove that merely functional disorder exists, for they by no means accompany invariably structural lesions.

It has been seen that functional disorder has certain pathological relations. The presence of the morbid conditions in connection with which it is apt to occur, is to be taken into account with the diagnosis. Thus, cardiac disturbance is presumably functional, if it be connected with plethora, anæmia, derangement of the nervous system from excessive venery, mental anxiety, the use of tobacco, dyspepsia, or gout. On the other hand, organic disease, in a large proportion of cases, originates in acute rheumatism. Hence, if a patient have never had the latter affection, this fact increases the chances that the cardiac disorder is merely functional.

The age of the patient is to be considered. Functional disorder occurs especially in the young, or between the age of puberty and middle life. Organic disease is oftener presented during or after the middle period of life. Functional disorder is oftener met with in females than in males; the reverse being true of organic disease. Organic disease occurs more frequently among the laboring classes of society, especially those exposed to the vicissitudes of the weather; functional disorder is more common among the sedentary and luxurious.

Disturbed action from functional disorder is apt to occur especially at night, probably because the mind of the patient being abstracted from outward objects, the attention is more likely at this time to be directed to the heart, or the thoughts are more self-concentrated. Persons with organic disease experience more inconvenience during the daytime, when they are exposed to causes which excite the circulation, such as exercise. Disturbance of the heart's action beyond that which is habitual, in persons affected with organic disease, is generally proportionate to obvious exciting causes. On the other hand, the action of the heart in cases of functional disorder is often out of proportion to appreciable causes; a sudden start, for example, sometimes occasions violent palpitation. Severe paroxysms of functional disorder often are not attributable to any apparent exciting cause.

The foregoing points are to be considered in the discrimination of functional disorder from organic disease; but, singly or collectively, they are never sufficiently diagnostic to warrant a decision that organic disease is not present. A positive diagnosis demands the information to be derived from physical exploration. The latter affords the readiest as well as the only sure way of coming to a decision. The employment of physical exploration in cases of merely functional disorder is one of the most beautiful (if this expression may be allowed), as well as useful, of the practical applications of this method of examination. A few moments often suffice to decide that the heart is free from structural lesions; and, reasoning by way of exclusion, that the symptoms referable to the heart are consequently due to functional disorder only.

In excluding organic disease, the absence of physical signs referable to structural lesions is to be ascertained. Is the heart enlarged? This is to be determined by defining the boundaries of the superficial and deep cardiac regions by means which have been fully considered in Chapter I, and by ascertaining that the point of apex-beat is within the range of healthy variations. Does auscultation fail in detecting adventitious sounds or murmurs? This is almost, if not quite, enough to warrant the conclusion that valvular lesions do not exist. Are murmurs discovered? Then it is to be determined whether they are organic or inorganic. The differential points involved in this discrimination have been mentioned in another division of this chapter. The exclusion of organic disease is rendered more positive by ascertaining, not only the absence of the physical signs denoting structural lesions, but the normal character and relations, in all essential particulars, of the heart-sounds. These are to be observed in different situations, the auscultator interrogating, successively, the aortic, pulmonic, mitral, and tricuspid valves, in the manner already described.

But let it be assumed that organic disease is not excluded; in other words, that the signs of structural lesions are present. It is to be determined whether functional disorder be not superadded. This is to be done by comparing the amount of organic disease with the degree of disturbed action. If the latter be disproportionate to the former, it is probably due, in a great measure, to functional disorder dependent on other morbid conditions than the lesions of structure. The amount of organic diseases and the effects which are fairly attributable to them may be ascertained, approximatively, by means of the physical signs. Is the heart but little, if at all, enlarged, and do the heart-sounds preserve their normal characters and relations so far as to show that the lesions cannot involve, to much extent, obstructive or regurgitant effects; disturbed action, if excessive or considerable, is probably due mainly to superadded functional disorder. It is important, in this connection, to take into view the presence or absence of the morbid conditions which are likely to give rise to functional disorder, viz., plethora, anæmia, &c. The presence of these conditions adds much to the probability of the symptomatic phenomena referable to the heart being due to functional disorder. It is a common error to attribute all these phenomena to the lesions of structure, whenever the existence of the latter is determined—an error often unfortunate as regards the prognosis and treatment. The lesions may be innocuous, and the cardiac symptoms dependent altogether on coexisting functional disorder. It is to be borne in mind that structural lesions, as a rule, do not give rise to disturbance of the heart's action sufficiently to occasion much, if any, inconvenience, prior to enlargement of the organ; and not infrequently the organ becomes considerably enlarged before the attention of the patient is awakened to any symptoms denoting an abnormal condition of the heart.

The discrimination of cases of fatty degeneration of the heart from those of purely functional disorder is sometimes attended with difficulty. The difficulty arises from the fact that this form of organic disease does not present any positive physical signs. It is, therefore, not so easily excluded as are valvular affections and uncomplicated enlargement of the heart. In many instances, however, enlargement coexists with fatty degeneration, and not infrequently lesions of the valves are also conjoined. Exclusive of these complications, the symptoms referable to the heart in cases of fatty degeneration are analogous to those which denote functional disorder. This structural change occurs at a period of life when persons are not so much exposed to merely functional disorder as at an earlier age. The palpitation connected with it has not that violence which frequently characterizes disturbed action when the muscular structure is sound. The paroxysmal character of merely functional disorder is less marked. Feebleness of action, and perhaps irregularity, are permanent symptoms. These circumstances, taken in connection with the various events which have been noticed under the head of the pathological relations and effects of fatty degeneration, in Chapter II, will generally enable the physician to determine whether this affection be or be not present. But it is to be borne in mind that the morbid conditions giving rise to functional disorder may be associated with fatty degeneration, as well as with other varieties of cardiac lesion.

PROGNOSIS IN CASES OF FUNCTIONAL DISORDER OF THE HEART.

The prognosis in cases of functional disorder of the heart is always favorable. Although the irregularity and violence of the disturbed action are sometimes such as apparently to involve immediate danger, it is doubtful whether a paroxysm ever proved fatal; nor do any unpleasant results follow, except a certain amount of exhaustion and nervous excitement. Recovery from the morbid irritability of the organ may be expected, but it often tends to continue for a considerable length of time. Of this the physician should be aware, and it is well to forewarn the patient that the duration of his malady may be tedious. After being assured, however, that he is not affected with an organic disease, and finding, by experience, that paroxysms occur and pass off without accident or injury, he endures their recurrence with greater patience than at first, and, at length, if they be not severe, he comes to regard them with comparative indifference.

It was formerly supposed that functional disorder, if protracted, tends to eventuate in organic disease. This doctrine has been disproved by clinical experience. Changes of structure very rarely originate in disturbed action of the heart, however persisting. I have known persons who have suffered from attacks of palpitation, frequently repeated, for many years, without the heart becoming enlarged. In cases in which inordinate action has continued steadily for several successive months, the soundness of the organ has remained unimpaired, and complete recovery has taken place. It is pleasant, as well as useful, to be able to assure patients affected with functional disorder that they are not rendered thereby liable to organic disease.

Retaining in the second edition of this work the statements contained in the foregoing paragraph, it is to be added that long-continued increase, frequency, and force of the heart's action may lead to enlargement. This fact is exemplified by some cases of exophthalmic goitre or Graves's disease, and also by cases in which overaction of the heart has been long kept up by active muscular exercise.[1]

TREATMENT OF FUNCTIONAL DISORDER OF THE HEART.

Therapeutical indications in cases of functional disorder of the heart, relate to two objects, viz.: *First.* Relief of disturbed action when present. This object embraces palliative measures only. *Second.* Removal of the morbid irritability of the organ. This object embraces curative measures, in other words, those by which it is expected recovery will be effected.

Curative indications are derived chiefly from the pathological relations and causes of functional disorder. The abnormal conditions with which morbid irritability of the heart is connected being different in different cases, the treatment cannot, of course, be uniform. The measures of therapeutics, in fact, differ, in individual cases, not less than the conditions on which cardiac disorder is dependent.

When associated with plethora, depletory measures are indicated. Bloodletting, locally or generally, may be judicious in some cases. It should, however, be employed with circumspection. Resorted to when not indicated, or carried too far, it tends to aggravate the cardiac disorder. This is shown by the effect of hemorrhages, and of the injudicious employment of bloodletting, formerly more than now, in various affections. The "reaction from loss of blood," as illustrated by the researches of Marshall Hall, and others, expresses phenomena which are mainly due to abnormal irritability of the heart. The existence of plethora is to be clearly ascertained before resorting to bloodletting, and it is to be borne in mind that the cases in which this condition of the blood exists are comparatively few. In most cases, if the existence of plethora be sufficiently evident, an adequate amount of depletion may be received by saline laxatives and by diet. The latter methods of depletion must not be pushed too far, or continued too long. The limit is the restoration of a normal condition of the blood. If the proportion of red globules be reduced below that of health, there is risk of the cardiac disorder being increased rather than diminished. When the proper limit is reached, habits of active exercise are to be conjoined with a nutritious, but not over-generous, diet. Animal food should be taken sparingly, and alcoholic stimulants avoided. These are the measures indicated by the coexistence of plethora.

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[1] *Vide* Chapter I, p. 30.

Associated with anæmia, which is vastly more frequent, the measures indicated are the reverse of those appropriate when plethora exists. The treatment now should be directed with a view to increase the proportionate quantity of the red globules of the blood. For this end, tonic remedies, and especially preparations of iron, are to be employed. The diet should be highly nutritious, and consist of a good proportion of animal food. Alcoholic stimulants, in the form of spirits, wine, beer, or porter, are generally useful. Moderate exercise in the open air is to be enjoined. The causes which have induced, and which may perpetuate the anæmic condition, are to be ascertained, and, if possible, removed. This will embrace the appropriate treatment of various local affections which in females are often seated in the genito-urinary system, such as leucorrhœa, menorrhagia, &c.; weaning in certain cases, avoidance of pregnancy, and, in short, proper attention to all the various circumstances which, in different cases, may be involved in the production and continuance of the anæmia. Bloodletting and other measures of depletion, in cases belonging to this class, are positively pernicious, and may prove so in a marked degree. The discrimination of these cases from those in which the cardiac disorder is connected with plethora, is highly important with reference to appropriate treatment. The fact that in the great majority of instances functional disorder of the heart is more or less dependent on anæmia is not to be lost sight of.

Derangements of the nervous system, arising from different causes, can only be treated successfully when the latter are ascertained and removed. Until these ends are attained, the cardiac disorder will be likely to continue. The general indication, in the cases coming under this head, is to place the patient without the influence of certain morbid agencies. The most prominent of these are, sexual excesses, the abuse of tobacco, tea, or coffee, excessive mental exertion, vigilance, and mental anxiety from a variety of causes, either real or imaginary. Curative measures consist in removing these causes, so far as they are controllable, together with the employment of remedies and a regimen calculated to restore the healthy condition of the nervous system. Change of scene, the excitement of travel, and recreation, are often highly useful, and may be sufficient to effect recovery when the disorder depends mainly on causes pertaining to the mind, as is not unusual. The importance of inquiry with respect to sexual excesses, is to be borne in mind. I have met repeatedly with cases in which cardiac disorder was traceable to this source. As already remarked, it appears to be an impression with some persons that indulgence cannot be excessive except when it is meretricious; hence, it is not enough to know that a patient is married. Under the head of sexual excesses, self-pollution is included. Of the difficulty often in obtaining information concerning this matter, especially with regard to females, it is unnecessary to speak. This may account for the obstinacy with which functional disorder of the heart persists in certain cases.

The coexistence of dyspeptic ailments calls for a proper regulation of diet and regimen, together with remedies to relieve gastric derangements and improve digestion. So far as the cardiac disorder depends on functional disturbance of the stomach, the treatment resolves itself into that due to the latter, of which the former is but a symptom. To consider the treatment of dyspepsia, would be here out of place. It may, however, be remarked that the ailments comprehended by this term generally involve morbid conditions seated elsewhere than in the affected organ, and often depend on mental causes. The treatment, therefore, must have reference to these ulterior conditions. In the dietetic management, it may be added, the object is not to reduce the diet to an extent corresponding to the weakened digestive power, but to invigorate and strengthen the latter, so that ordinary wholesome articles of food may be taken without inconvenience. The measures for this end are, tonics, stimulants, exercise in the open air, mental recreation, and persistency in a nutritious and varied diet in spite of occasional symptoms denoting difficult or imperfect digestion. It is mistaken policy to watch the effects of taking particular articles of food, and to eliminate, successively, from the diet, those which are found to occasion inconvenience. Various accidental causes may disturb digestion, and a meal which on one day may be followed by distress, on the next day may be taken without trouble. The practical rule for the dyspeptic, as regards diet, is to eat the ordinary, wholesome, well-cooked varieties of food in sufficient quantity to meet the wants of the system, trusting to remedies and regimen to render the digestive organs adequate to the performance of their duty. These remarks are, of course, only applicable to cases of merely functional disturbance of the stomach.

Functional disorder of the heart occurring in persons affected or threatened with gout, claims the remedies which are indicated by the gouty diathesis. These are medicines supposed to act by elimination, of which the most efficient is colchicum, and alkalies given with a view to neutralize the excess of lithic acid in the blood. Of the latter, potash is considered as preferable to soda, in consequence of the solubility of the salt formed by the union of lithic acid with the former. The salts of lithia are especially useful, the lithia combining with the lithic acid and forming a compound which is readily eliminated.

The several morbid conditions which give rise to functional disorder of the heart may be more or less combined in certain cases. Under these circumstances, the treatment must have reference, of course, to the different conditions existing in combination. The gouty diathesis, for example, may be conjoined with plethora, or dyspepsia; dyspepsia and anæmia are often united, &c. It is not enough to have ascertained the existence of one of the conditions upon which cardiac disorder may be dependent; the inquiry is to be extended so as to embrace others which may coexist.

One of the most important of the means of promoting recovery is applicable to cases occurring in each and all of the different pathological connections. This is the moral influence of a positive assurance that the heart is free from organic disease. The anxiety and apprehension incident generally to disturbance of the heart's action, tend powerfully to perpetuate and aggravate the disorder. If the physician be sufficiently confident in his diagnostic ability to assure the patient confidently that the affection is purely functional or inorganic, and the patient have sufficient confidence in the knowledge and judgment of the physician to believe the assurance, this will often go very far toward promoting a cure. The effect, in many instances, is truly remarkable. Hence, a great practical advantage is to be derived from a sure diagnosis. An opposite effect is equally marked when the patient is told that he has organic disease. I have met with instances in which several years were embittered by a false diagnosis and its imprudent communication. This, with the measures generally advised in conjunction, suffices to perpetuate the disorder indefinitely. If the physician be competent to employ physical exploration, and to satisfy himself thereby that organic disease does not exist, he should take pains to remove the groundless fears of the patient, at the same time forewarning him that the disorder is liable, when it once occurs, to continue for a greater or less period. If the patient can be made to believe that there is no danger, the malady is rendered supportable until recovery is effected. This is a point in the treatment of functional disorder of the heart, second to none other. In all cases the patient should be advised not to watch the action of the heart by feeling the pulse or the apex-beats, or listening to the sounds at night. His attention should be diverted from the disordered organ as much as possible. The benefit of agreeable occupation is, in part, explicable by its effect in this way.

Functional disorder, not dependent on, but coexisting with, organic disease, claims essentially, the same measures as when disconnected from the latter. With reference to treatment, it is highly important to determine that functional disorder from some one or more of the morbid conditions which give rise to it is superadded to organic disease. I have met with instances repeatedly in which, in consequence of this combination, patients appeared, at first view, to be in an advanced stage of organic disease, but who recovered, by judicious management, apparently, perfect health. The practitioner cannot be too often cautioned not to attribute all the symptomatic phenomena referable to the heart to structural lesions, when the latter are found to exist. The practical rule may be here repeated, to regard these phenomena as probably due to functional disorder whenever the heart is but little, if at all, enlarged. The association of anæmia with a certain amount of organic disease is quite common, when, if the blood be restored to its normal condition, the cardiac lesions are found to be, for a time at least, innocuous. But if the lesions involve more or less obstruction or regurgitation, and enlargement of the heart have already taken place, marked improvement, as regards the cardiac and other symptoms, may be expected to follow the removal of the conditions which give rise to the associated functional disorder.

The foregoing remarks have had reference to measures which are distinguished as curative. Palliative measures are now to be considered. These are to be adapted to different circumstances pertaining to the disturbed action of the heart To tranquillize the excited organ and restore regularity of action, are the ends for which palliative measures are pursued. But the heart is subject, as has been seen, to various forms of disorder, and the condition of the organ, as manifested by the symptoms, is not precisely the same in all the several varieties. In the mildest form of disorder, in which only an occasional, momentary disturbance is felt, there is neither necessity nor time for palliation. Curative measures are alone required. Persisting, inordinate action, continuing perhaps for weeks or even months, calls for remedies calculated to allay this state of excitement. Clinical observation appears to show that digitalis is not an appropriate remedy in these cases. This remedy is especially useful when weakness and irregularity of the heart's action are incident to cardiac lesions. Aconite is more appropriate, given in small doses repeated after short intervals. Hydrocyanic acid, or the laurel water, hyoscyamus, belladonna, and other narcotic sedatives, may be employed in succession. A belladonna plaster worn over the præcordial region appears frequently to exert a happy effect. Opium is admissible, in some cases, bearing in mind the risk of becoming accustomed to its use, and the formation of a habit which is with difficulty broken, and which entails evils of no small magnitude. Palliative means in these as in other cases, are, of course, to be conjoined with measures which are designed to be curative.

Paroxysms of palpitation characterized by violence and irregularity of the heart's action, may be shortened and mitigated by palliative measures. Cases of functional disorder often come under the observation of the physician, for the first time, under these circumstances. He is frequently summoned in haste, and finds, perhaps, the patient and friends in a state of great alarm. The first point is to give assurance of absence of danger, as soon as it is ascertained that the disturbance is merely functional. A full opiate affords often the quickest and most reliable method of procuring relief. Revulsive applications are serviceable, such as sinapisms to the chest, or, compresses moistened with strong aqua ammoniæ. These may be applied over the spine if there be tenderness on pressure. Warm, stimulating foot-baths are highly useful. These measures relieve by obviating the tendency to the accumulation of blood within the heart. This tendency is shown by coldness of the surface and extremities during paroxysms of palpitation. Painful stimulation of the surface is also useful by diverting the attention of the patient from the heart. If an opiate be not employed, the remedies called antispasmodic are indicated, such as the ethereal preparations, the compound spirits of lavender, the aromatic spirit of ammonia, valerian, assafœtida, &c. Some of these may be given in conjunction with, or in addition to, opium or the salts of morphia.

In paroxysms characterized by feebleness of the heart's action, with intermittency and a tendency to syncope, prompt relief is often afforded by alcoholic stimulants. Brandy or some form of spirit should be given pretty freely, and not much diluted. Anodynes, antispasmodics, and revulsive applications may be added.

The symptoms associated with palpitation, exclusive of those referable to the heart, must influence, to some extent, palliative measures. Thus, if plethora be manifestly present, a small venesection or local bloodletting by cupping or leeching, may be indicated with a view to immediate relief. Marked coldness of the surface, on the other hand, and prostration, point to the free use of stimulants. Remedies addressed to the stomach, in certain cases, are effectual. If the stomach be distended with gas, carminatives act like a charm. If acidity or cardialgia be present, an alkaline or antacid remedy, and especially ammonia, may cut short the paroxysm. If the bowels be constipated and flatulent, an active cathartic, or a large, stimulating enema may prove equally efficient.

In paroxysms occurring in persons of a gouty habit, it has been advised to make irritating applications to the joints usually affected, in order to solicit the local manifestations of the disease in these situations. Preparations of colchicum and guaiacum are considered as indicated by palpitation occurring under these circumstances. The palliative measures, however, suited to other cases are applicable, and especially remedies addressed to the stomach.

CHAPTER X.

DISEASES OF THE AORTA.—THORACIC ANEURISMS.

Acute aortitis—Subacute or chronic aortitis—Morbid deposit on the surface of the lining membrane of the aorta—Atheroma—Calcareous deposit—Dilatation of the aorta—Contraction and obliteration of the aorta—Thoracic aneurisms—Definition—Varieties—Anatomical relations, &c.—Causes—Terminations—Symptoms—Physical signs—Diagnosis—Treatment.

AFFECTIONS of the aorta do not, strictly, fall within the scope of a treatise on the diseases of the heart; but they are with propriety included, not merely on account of the close anatomical relations of the parts affected, but because diseases in these two situations are often associated, and, without proper knowledge and care, certain symptoms and signs due to aortic affections, are liable to be attributed to the heart. The aorta may be the seat of inflammation, acute and chronic, and of structural changes either resulting from inflammation or non-inflammatory; it is subject to alterations in calibre, viz., contraction or dilatation, and aneurisms occur oftener in this than in any other portion of the arterial system. This chapter will be devoted to a brief consideration of these several forms of disease, treating of them only so far as they are of practical interest to the physician.

Acute inflammation of the aorta is one of the most infrequent of diseases. It might naturally be presumed that inflammation would be likely to extend into this artery, from the left ventricle, in endocarditis, but it is quite otherwise; the latter disease is sufficiently common, and in the few cases of acute aortitis which have been observed, it does not appear to have been always either preceded or accompanied by endocardial inflammation. There are various pathological topics pertaining to inflammation within the aorta, which have been much discussed, and are still open for discussion, such as, the primary seat of the inflammation, the occurrence, or not, of fibrinous exudation on the free surface of the lining membrane, the production of pus and consequent purulent infection of the blood, the coagulation of fibrin within the vessel, &c. I do not propose to enter into a discussion of these topics. They possess pathological interest and importance, but they are not embraced within the practical objects of this work. As regards acute aortitis, with our present knowledge of its symptoms, signs, and pathological effects, no physician would venture to diagnosticate it. This fact furnishes a sufficient reason for not devoting to it distinct consideration.

The statement just made respecting acute inflammation of the aorta, may also be applied to subacute and chronic aortitis, so far as concerns symptoms, signs, and pathological effects, referable immediately to the inflammatory state. In a practical point of view, certain degenerative changes and lesions of the aorta are chiefly important. It is a pathological question how far these are the ulterior consequences of inflammation. This question involves the latitude of signification belonging to the term inflammation; and there would be no advantage, practically, in here discussing it. The important degenerative changes are those commonly known as atheromatous and calcareous. To these I shall give brief consideration. Next dilatation, contraction, and obliteration of the aorta, will claim notice; and, lastly, aortic aneurisms will require to be considered at some length with reference, especially, to the diagnosis and treatment.

ATHEROMATOUS AND CALCAREOUS DEGENERATION OF THE AORTA.

The changes embraced under the name *atheroma* are very common in the aorta. They exist, more or less, in most persons after middle age. The earliest appearance is that of small white spots. Increasing and coalescing patches, irregular in form, and of greater or less size, are formed. They are due to the presence of a morbid product, of a pulpy consistence, as the name atheroma denotes. This atheromatous product is found, on microscopical examination, to contain oil globules in abundance, with crystals of cholesterine; and hence, it is considered as a variety of fatty degeneration. It is situated beneath, not upon, the free surface of the lining membrane of the artery. At first, a semisolid, it may undergo softening and become a puriform liquid. The lining membrane of the artery, after a time, gives way, either from pressure or molecular changes, and the liquefied product is discharged, leaving ulcerated and sometimes cyst-like spaces. The weakness of the vessel at these points may lead to aneurism, and even to perforation; the latter, however, is extremely rare. According to Virchow, and others, the atheromatous product is the result of a granulo-fatty transformation of the anatomical elements of the part, these having been increased by proliferation, and the primary morbid process is inflammatory.

The calcareous degeneration appears to have its point of departure in atheroma. Instead of becoming liquefied and being discharged, the atheromatous product is solidified by the deposition of the phosphate, and carbonate of lime. It undergoes calcification. This degenerative process commences beneath the free surface of the lining membrane; but, after a time, the lining membrane is destroyed, and the calcareous matter, in the form of plates or scales, projects within the artery, and is in contact with the current of blood. The amount of the atheromatous and calcareous degenerative changes varies greatly in different cases. Both forms of degeneration may be combined. The portions of the artery affected may be few and small, or numerous and large. They may be confined to a limited section of the ascending part of the arch, or they may extend over a great part or the whole of the artery.

The atheromatous and the calcareous degeneration stand in a causative relation to the aortic lesions which remain to be considered in this chapter. There are, also, other pathological effects. The roughness of the inner surface of the artery is apt to lead to deposits of fibrin, and these being detached form emboli. Hence may arise the local evils dependent on embolism of the arteries of the brain and in other situations. It is probable that toxæmic effects, which, with our present knowledge, are undefined, may arise from the admixture of the liquefied atheromatous matter with the blood; moreover, with this matter more or less pus may be combined. The loss of the elasticity of the aorta, when it is extensively affected with either form of degeneration, and the resistance which it offers to distension from the pressure of the blood, if it be greatly calcified, are other effects. The latter may give rise to hypertrophy of the left ventricle, which, however, under these circumstances, is compensatory or consecutive. Weakness of the circulation is incident to each of these effects in proportion as the force of the recoil of the arterial coats, which follows the ventricular systole, is impaired.

There are no subjective symptoms which point, with any distinctness, to atheromatous or calcareous degeneration of the aorta. The atheromatous condition may not give rise to any physical sign; but calcareous deposits projecting within the artery will cause a systolic murmur, which may be loud or feeble, and soft or rough, according to the force with which the left ventricle contracts, and the physical conditions within the artery. How is this murmur to be distinguished from a systolic murmur produced at the aortic orifice? The only points of difference are, the maximum of the intensity of a murmur produced within the artery is likely to be higher, that is, at a greater distance above the base of the heart, and the murmur is less likely to be loud or appreciable below the second intercostal space. The absence of an aortic regurgitant murmur, and the intensity of the aortic second sound, showing integrity of the semilunar valves of the aorta, are negative points bearing on the discrimination. A short murmur following the systolic, and ceasing with the second sound, which, in a former part of this work, I have called a pre-diastolic murmur,[1] is a distinctive sign which, in some cases, can be very clearly made out. Its significance in this point of view renders it important. If the arterial murmurs be associated with lesions of the aortic orifice giving rise to an aortic direct and an aortic regurgitant murmur, the diagnosis is difficult or impossible. An inorganic arterial murmur is to be excluded; and it is to be borne in mind that this murmur may be, although it rarely is, intense and rough. Aortic aneurism and dilatation of the aorta are to be excluded. It may be here stated that a murmur in the aorta, however intense or rough, is never alone evidence of aneurism. The age of the patient is to be considered. The atheromatous and the calcareous degeneration occur chiefly after middle age, and especially in the aged; hence, they are called senile alterations of the arteries.

Marey has pointed out certain characters of the pulse, as delineated by the sphygmograph, which belong to senile alterations of the arteries. In the figure representing the form of the pulse, the line of ascent is vertical, showing quickness of the pulsation;

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[1] *Vide* p. 213.

the summit is obtuse, or presents a horizontal line, and the line of descent shows the absence of dicrotism.[1]

The treatment of the atheromatous and calcareous degeneration of the aorta does not claim consideration. Practically, the advantage of taking cognizance of them consists in avoiding the error of attributing the murmurs which are incident to them, together with the characters of the pulse, to lesions at the aortic orifice of the heart.

DILATATION OF THE AORTA.

Dilatation of the ascending aorta is a not infrequent result of atheromatous and calcareous disease. The middle and lining tunics becoming softened, attenuated, and the elasticity of the vessel impaired or lost, dilatation takes place from the distending force of the blood-current propelled forward by the systole of the left ventricle, and backward by the recoil of the arterial coats beyond the affected portion of the aorta. This result is more apt to follow if the left ventricle become hypertrophied. The ascending portion of the arch and the sinuses of Valsalva are the points most apt to yield to the distension. In the latter situation, the dilatation is probably produced more by the retrograde than the onward current, provided the semilunar valves remain sufficient. The aorta may be dilated to double or treble its normal size, and cases have been reported of a much larger increase. Dilatation is distinguished from aneurism by the absence of the sacculated forms which characterize the latter; but not infrequently aneurism and dilatation are associated. In some cases of dilatation the walls of the arteries appear not to have been previously the seat of disease. The walls of the dilated portion are sometimes thickened, and sometimes attenuated. The attenuation has existed to such a degree as to lead to rupture. Coagulated fibrin never accumulates in the dilated artery as it does in an aneurismal sac; but if the lining membrane be

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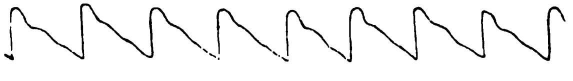
[1] The following, from Marey's work, represents the characters of the pulse in a patient, 52 years of age, who died of cerebral hemorrhage, the autopsy showing notable calcification of the aorta.



roughened with calcareous deposits, these may serve as nuclei for fibrinous masses which may become detached and form migratory plugs or emboli.

To make out, during life, the existence of dilatation when it is not extremely great, is certainly a very difficult problem; and, if the dilatation be extremely great, to discriminate between it and aneurism is difficult. According to Bellingham, visible pulsation of the large arteries of the neck and upper extremities, and a jerking or receding pulse, are characteristic symptoms. These have been noticed in a previous chapter as distinctive of lesions at the aortic orifice permitting regurgitation from the artery into the left ventricle. Their significance as denoting dilatation of the aorta, disconnected from aortic insufficiency, must depend on the presence of adequate evidence that the semilunar valves remain sufficient. This evidence consists in the absence of a murmur denoting regurgitation into the ventricle, and the intensity of the aortic second sound of the heart being but little, if at all, impaired. It is only under these conditions that these symptoms can be considered as indicating aortic dilatation. Bellingham also states that an impulse synchronous with the pulse is perceived when the ear is applied to the stethoscope laid upon the first bone of the sternum. This impulse may be perceived thus by the ear when it is not communicated to the hand with sufficient force to be appreciable. In connection with these signs, a double rough murmur is perceived which is referable to the aorta and not to the aortic orifice. The diagnosis in some cases, as claimed by the author just named, may be made out by means of this combination of physical signs; but in most cases, the diagnostic points are invalidated by the coexistence of lesions at the aortic orifice.

CONTRACTION AND OBLITERATION OF THE AORTA.

The calibre of the aorta is sometimes more or less diminished in consequence of atheromatous and calcareous degeneration; also, by vegetations and polypoid growths. The deposit of coagulated fibrin in connection with the foregoing changes, or the production of thrombi, is another cause. Contraction from these intrinsic causes affects generally the ascending portion of the aorta, and it is difficult to distinguish it, during life, from obstruction produced by lesions at the aortic orifice. Lesions at the aortic orifice, either obstructive or regurgitant, or both, are generally associated. Extrinsic causes may produce contraction of the aorta. Aneurismal and other tumors are sometimes so situated as to have this effect. The diagnosis of aortic contraction, under these circumstances, can only be conjectural. Again, contraction, and even obliteration of the aorta have been repeatedly observed at or near the junction of the ductus arteriosus. In this situation the diagnosis may sometimes be made with much positiveness.

All the cases of contraction and obliteration of the aorta near the junction of the ductus arteriosus, which had been reported up to 1860, 40 in number, have been collected and analyzed by Dr. Thomas B. Peacock, and an abridgment of his analysis will be here given.[1]

The contraction may be abrupt, and limited to a small section of the vessel; or the aorta may gradually diminish in size, a large portion being more or less contracted. In 1 of the 40 cases the aorta was smaller than usual throughout its whole extent. When abrupt, as it was in 22 of the 40 cases, it is generally described as appearing to have been produced by a knife pressed on the coats of the vessel, or a string tied tightly around it, or a ring passed along it. The constriction was often greater than appeared externally, due to contraction and thickening of the internal tunics. In 8 of the cases the calibre was obstructed by a septum formed by a duplicature of the internal coats, which in one case completely closed the canal, and in other cases left a small triangular, oval, or rounded aperture. In 2 cases the septum had a bi-concave form; in 1 case it presented two projecting lips, and in 1 case it was protruded forward, forming a funnel-shaped aperture. In 10 cases the canal of the vessel was entirely obliterated, and in the remaining 30 cases the degree of contraction varied, the canal admitting only a small flat or blunt probe in 8 cases, a straw in 1 case, and a writing-quill in 3 cases.

The condition of the vessel above and below the point of contraction or obliteration varied. Generally the ascending portion was dilated, the coats frequently being thickened with atheromatous or calcareous deposit. In some cases these changes extended over a large part of the arch, and even to near the seat of the contraction. In 11 cases, however, dilatation alone existed, the coats not appearing to have been materially diseased. Below the seat of the stricture generally the vessel was quickly dilated, but in some cases the size of the vessel was smaller than natural.

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[1] For further details of the cases and an account of the previous literature of the subject, see Dr. Peacock's paper in the British and Foreign Medico-Chirurg. Review, April, 1860.

That the contraction or obliteration of the aorta, in these cases, involved a congenital imperfection, is probable, although in the larger number of cases, namely, in 24 of 38 cases, the ages were between twenty-one and fifty years. In the remainder of the 38 cases in which the ages were noted, they varied from twenty-two days to twenty years. The occurrence in a considerable number of cases under the age when the ordinary affections of the aorta are most apt to occur, the situation of the obstruction near the junction of the ductus arteriosus, and the fact that the coats of the vessel are not always diseased, are valid grounds for referring the origin to an error, at birth, in conformation. This view, which writers have generally held, is corroborated by the frequency of other congenital defects in the cases analyzed. Thus, of the 40 cases, in 5 the aortic semilunar valves were defective; in 1 case the columnæ carneæ of the mitral valves were wanting; in 1 case the septum of the ventricles was imperfect; in 1 case the foramen ovale was unclosed; in 3 cases the ductus arteriosus was more or less pervious throughout its whole extent; and in 4 cases it remained open for a portion of its aortic extremity, but was obliterated at the pulmonic end; in 1 case there was hare-lip, with fissure of the palate; in 1 case there was hypospadias; and in 2 cases there was defective conformation of the lower extremities. The aggregate of these cases is 19, nearly one-half the whole number, a proportion certainly greater than would be met with in an equal number of cases of any form of disease not congenital. The congenital defect, however, may increase during the progress of life, and it is aggravated by diseases developed after birth.

The maintenance of life, and even of vigorous health, for many years, when the aorta is greatly contracted, and even obliterated, is remarkable as illustrating the ability of collateral vessels in carrying on the circulation. From an examination of bodies in which the arteries were injected previous to dissection, it appears that the branches of the subclavian arteries, and the aortic branches below the seat of the obstruction are channels of communication adequate to a sufficient supply of blood to the lower parts of the body.

The branches referred to, are, 1st, the transversalis colli, derived from the thyroid axis, which, through its posterior scapular branch, anastomoses with the posterior branch of the aortic intercostal arteries; 2d. The superior intercostal artery, derived from the subclavian, which anastomoses with the aortic intercostals; and, 3d. The internal mammary, which, by means of its anterior intercostal branches, communicates with the anterior aortic intercostal artery, by its musculo-phrenic artery unites with the lower intercostals, and, by the superior epigastric artery, with the epigastric derived from the external iliac artery. The thoracic branches of the axillary artery appear also to assist in conveying the blood into the descending aorta. All these vessels are found to have become very greatly enlarged, elongated, and tortuous.

The obstacle to the circulation, however, produces the same effects upon the heart as other forms of aortic obstruction, namely, hypertrophy and dilatation. In 28 cases there was more or less enlargement of the heart, the enlargement being confined to the left ventricle in 10 cases, and the right ventricle being also affected in 18 cases. Aortic valvular lesions coexisted in 12 cases, and mitral valvular disease in 1 case. In several of the cases of aortic lesions the valves were simply incompetent from dilatation of the artery at its orifice.

Of the 40 cases, in 12 there was no history of the symptoms during life, the patients dying suddenly, or of some disease not connected with the condition of the aorta, and the defect was only detected on examination after death. In 6 cases the patients came under observation for some acute or chronic affection of an independent character, and the evidences of some defect in the vascular system were detected, as it were, accidentally. In 18 cases the patients, after having enjoyed good health and been equal to active exertion for a long period, manifested the symptoms of cardiac disease, such symptoms often appearing after some obvious exciting cause, and gradually advancing until they assumed the usual features of cardiac asthma and dropsy. In these cases the interval which elapses between the first occurrence of the symptoms, and the fatal termination varied greatly, in some cases being one, two, three, or four months; in other cases, five and eight years. In 4 cases only had the patients always been delicate, manifesting especially symptoms of feebleness of the circulation, such as breathlessness, lividity, chronic cough, &c.

As regards the causes which immediately occasioned death, in 11 cases the patients died of acute or chronic diseases but little, or only indirectly, connected with the morbid condition of the vascular system. In 8 or 9 cases death occurred suddenly, and was directly traceable to the condition of the aorta. In 3 cases, blood escaped into the cavity of the pericardium from rupture of recent dissecting aneurisms of the ascending aorta, and in 1 case from a dissecting aneurism of the arch; in 1 case the right ventricle was the seat of rupture, and in 1 case, the right auricle; in 1 case a large aneurism of the descending aorta, below the seat of the constriction, had burst into the left bronchus, and in 1 case, death occurred from syncope attributable to fatty degeneration of the right ventricle. In 1 case the patient was reported to have died instantly from apoplexy, and in this case, probably, the death was from syncope. In the largest proportion of cases, namely, in 16 of 36, in which the cause of death was clearly stated, the patients sank with the ordinary symptoms of cardiac asthma and dropsy, complicated, in different cases, by bronchitis, pneumonia, pleurisy, pericarditis, erysipelas and sloughing, purpura, &c.

The diagnosis of contraction or obliteration of the aorta near the junction of the ductus arteriosus has been made by Oppolzer, Hamernjk, and Van Leuwen, and its correctness verified by examination after death. The diagnosis was based on the following points: 1st. Notable enlargement, contortion, and pulsation of collateral branches of vessels originating from the arch of the aorta, namely, the transversalis colli, the posterior scapular, and the superior aortic intercostal arteries, together with the superficial branches distributed to the chest and back. 2d. Notable weakness of the pulsation of the arteries of the lower extremities, as the inguinal and the femoral. This contrast between the pulsation of the arteries of the upper and the lower parts of the body is, of course, the more striking according to the degree of the aortic obstruction, and is most marked when the canal of the vessel is entirely occluded. 3d. In some cases a systolic murmur, most intense at the base of the heart, along the upper part of the sternum, under the clavicles, especially on the left side, and at each side of the neck in front. In the case observed by Hamernjk a murmur was audible in the dilated superficial arteries.

These points indicate an obstruction below the origin of the left subclavian artery as the situation of the aortic affection. But to arrive at a diagnosis of contraction or obliteration of the aorta, obstruction from the pressure of an aneurismal or other tumor must be excluded; there must, therefore, be no deficiency of resonance on percussion, no evidence of pressure on the recurrent laryngeal nerve, the œsophagus, trachea, or bronchi.

The affection is apt to be overlooked, because in some cases no marked symptoms of disorder of the vascular system are manifested during life; and in the cases in which obvious symptoms are produced, they are often due to the secondary affection of the heart rather than to the original difficulty, and do not appear to differ in any material respect from those of cardiac disease dependent on any other source of aortic obstruction.

THORACIC ANEURISMS.

The term aneurism, in its broadest sense, and in accordance with its etymology,[1] is applicable to every species of dilatation of an arterial trunk. It is customary, however, to exclude dilatations which are not circumscribed, and which are not pouch-like in form. The term is also applied to a sac or pouch communicating with an artery through a perforation caused either by disease or a wound. When the perforation is caused by a wound, the aneurism is distinguished as traumatic, and all aneurisms produced by disease are called spontaneous. Sometimes, after the rupture or wound of an artery, the blood, instead of being contained in a sac, infiltrates the surrounding tissues, over a considerable or large space; the aneurism is then said to be diffused. The propriety of applying the term aneurism to the infiltration of blood after perforation of an artery, has, with justice, been denied.

The division into *true* and *false* aneurisms has been long maintained, in the former the artery being dilated without solution of continuity of the coats, in the latter the coats having been ruptured or destroyed over a certain space. Aneurisms are said to be *mixed* when they arise from dilatation of one or two of the coats, the other coat or coats having been ruptured. Mixed aneurisms have been called *external* when the sac is formed of the outer coat, the internal and middle coat having been ruptured; and they have been called *internal* when the sac is formed by the internal coat protruding through the ruptured middle and outer coats. The latter is extremely rare. An aneurism may be at first true, and afterward become mixed, or it may be primarily an external mixed aneurism; the latter obtains in the great majority of cases. The tumor formed by an aneurismal sac is generally smooth and globular, but it is sometimes oval or conical in shape, and it may be rendered irregular by secondary and even tertiary dilatations giving it a mulberry appearance externally, and causing it to present internally a multilocular arrangement. These variations have given rise to other divisions. A variety is called the *dissecting* aneurism. In this kind, rupture of the inner and middle coats of the artery first occurs, and the blood, instead of dilating the outer coat so as to form a sac, detaches this coat over a greater or less distance. In an instance reported by Pennock,[1] the dissection of the coats extended as far as the primitive iliacs. The aorta in this case presented the appearance of a double cylinder, that situated internally being the aorta proper, communicating directly with the heart, and surrounding this a much larger cylinder communicating with the inner one by a valvular fissure half an inch in length. In some instances the blood which separates the coats, after passing a certain distance, finds its way again into the proper arterial channel through a second opening.

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[1] *ἀνεύρυσμα*, a dilatation.

Finally, the names varicose or cirsoid aneurism and aneurismal varix are applied to a lesion in which a communication has been established between an artery and a vein. I agree with my friend Prof. Gross in the opinion that the so-called dissecting aneurism and the aneurismal varix do not come within the proper definition of an aneurism, and that they should not be reckoned as such.

An account of each of the various kinds of aneurism should, of course, enter into a full consideration of the subject; but, directing attention exclusively to aneurisms of the thoracic aorta, and considering these only so far as they are of practical interest, reference will be had in this chapter chiefly to sacculated enlargements, as distinguished from dilatations of the artery, constituting either true or mixed aneurisms. With reference to diagnostic phenomena, certain points are to be noticed prior to considering the symptoms and physical signs.

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[1] Note in Hope on Diseases of the Heart, 1842, p. 402. Several cases are here reported by the editor, Dr. Pennock. Similar cases have been described by numerous other writers.

Sacculated thoracic aneurisms form tumors varying in size from a pea to the fœtal head; but to give rise to phenomena available for diagnosis, the size must be considerable. Their situation determines, to a considerable extent, their symptoms and signs. They are seated oftenest in the ascending aorta, next in frequency at the arch, and less frequently in the descending aorta. Of 87 cases, in 40 they were connected with the ascending aorta, in 30 with the arch, and in 16 with the descending portion.[1] They occur sometimes at the sinuses behind the semilunar valves, and in this situation rupture takes place before the tumor attains to a large size, in consequence of the cellular coat which exists in other situations being here wanting and its place supplied by the more delicate pericardium. The effusion of blood, when rupture takes place, is into the pericardial sac, and death is usually produced almost immediately by mechanical compression of the heart. Aneurisms in this situation occur most frequently at the right coronary sinus; next at the posterior or inter-coronary sinus, and rarely at the left coronary sinus. Occurring at the right coronary sinus, the aneurismal tumor presses on the pulmonary artery, and may occasion more or less obstruction to the current of blood in that artery, thereby giving rise to enlargement of the right side of the heart. Regurgitation through the pulmonic valves is liable to be induced. A positive diagnosis of aneurisms, in this situation, is impracticable. In most cases there are no symptoms pointing to the existence of any disease of the heart or large vessels. In one of 4 cases which I have recorded, the patient fell and expired almost instantly while in the act of leaving hospital after recovery from pleurisy. In another case the person was found dead, having been up to the time of death apparently in perfect health. In another case sudden death took place, and in the fourth case rupture had not taken place. Situated above the valves, the aneurism may or may not be accompanied by dilatation of the artery at its orifice, so as to render the valves insufficient; or the aneurism may or may not be complicated with aortic valvular lesions. As regards the effects upon the heart, much depends on the existence or non-existence of aortic regurgitation. If regurgitation take place, enlargement of the heart, commencing and predominating in the left ventricle, is sure to follow; but if the aortic valves remain sufficient, the heart does not invariably become enlarged. Aneurisms of the ascending aorta and arch are generally seated on the convex side of the vessel, and the tumor extends in a direction to the right, the extension, of course, being proportionate to the size of the tumor. But it is to be borne in mind that the tumor may spring from either the concave or the posterior surface of the vessel and extend in different directions, having, consequently, different anatomical relations to the thoracic walls and the organs within the chest. Whatever may be the direction in which the tumor extends, in proportion to its size it presses upon surrounding parts, displacing them, and interfering with their functions; it gives rise to local inflammation and adhesions; it causes erosion and atrophy, and, finally, it ends frequently in rupture or an opening produced by sloughing or ulceration, through which the arterial blood escapes either externally or into some internal part. With reference to the relative liability of different portions of the thoracic aorta to aneurism, the following statistics are cited from Sibson's Medical Anatomy, Fasciculus V: "Of 703 cases, in 87 the aneurism was within the pericardium; in 193 the ascending aorta above the attachment of the pericardium was the seat; the transverse and ascending aorta were both involved in 140 cases; the transverse was alone affected in 120, and conjointly with the descending aorta in 20 cases; the descending portion of the arch was the seat in 72, and the aorta below the arch in 71 cases."[1]

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[1] Swett, op. cit., p. 544.

The anatomical relations of aneurismal tumors must be considered in order to understand the rationale of certain symptoms. Arising, in the majority of cases, from the convex margin of the ascending or transverse aorta, contracting adhesions with the thoracic walls, and erosion of the latter taking place, the integuments are at length raised, forming a visible swelling. This swelling or external tumor is generally situated at a point on the right side of the sternum between the clavicle and the fourth rib; but it may make its appearance over the first or second bone of the sternum, or in the neck, or below the left clavicle, and in rare instances on the posterior surface of the chest. But before it becomes apparent to the eye, certain effects are apt to be produced by pressure on internal parts. These effects are still more marked when the aneurismal tumor springs from the concave margin of the vessel. The parts exposed to pressure, and giving rise to symptomatic phenomena, are the trachea, the bronchi, the lungs, the œsophagus, the par vagum and recurrent nerves, the sympathetic nerve, the superior vena cava, the thoracic duct, and the pulmonary artery. The effects of pressure on these parts belong among the symptoms of thoracic aneurism. The anatomical relations of aneurismal tumors arising from the descending aorta are different. Pressure on the parts just mentioned does not occur. An external tumor is not developed unless the aneurism become extremely large. Aneurisms here situated are apt to contract adhesions to the dorsal vertebræ, leading to erosion of the bony structure, and sometimes an opening takes place into the spinal canal. Situated behind the heart, an aneurismal tumor may displace this organ, and, by pressing it forward, render its action on the thoracic walls so strong as to simulate hypertrophy.

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[1] For statistical facts relating to numerous points of interest and importance in the history of aneurisms, the reader is referred to Sibson's Medical Anatomy.

In addition to the size of aneurismal tumors, the portion of the aorta from which they spring, and the direction in which they extend, other points are concerned in the production of symptoms and signs. The mouth of the sac varies in size and form in different cases, allowing more or less freedom of the ingress and egress of blood, and either favoring, or otherwise, the force of the current into the cavity. The opening may be smooth, or roughened by calcareous deposit. The artery in the vicinity of the aneurism may be healthy, but oftener it is more or less diseased. The interior of the sac is frequently studded with calcareous plates. The cavity of the sac may be filled with liquid blood; but it often contains solidified fibrin in more or less abundance. This fibrin, deposited in a series of concentric layers, presents a stratified arrangement, the layers nearest the parietes of the sac being the most condensed, decolorized, and dry, and those in proximity to the blood softer, more moist, and reddened with hæmatin. The size of the cavity receiving blood is, of course, diminished in proportion to the accumulation of solidified fibrin. This deposit is favored by the large size of the sac, the smallness of its mouth, roughness of the interior surface, and feebleness of the heart's action. It must be considered as a conservative provision for strengthening the sac and retarding the progressive increase of the tumor. A spontaneous cure is sometimes effected by an accumulation of fibrin sufficient to obliterate the cavity. Masses of fibrin are liable to become detached from within the sac, forming emboli or plugs, which are carried onward with the current of blood, and, becoming impacted in arteries more or less remote, occcasion obstruction of the circulation, and a diminished supply of blood to important parts. Obstruction of arteries communicating with the portion of the aorta where the aneurism is seated, is an important effect. This arises from plugging of the arteries with fibrin, and from the small size of the mouth of the sac or the form of the aperture being such as not to allow free passage of blood into the cavity. It also proceeds from the outward pressure of the aneurismal tumor. The arteria innominata, carotid, and subclavian arteries are liable in this way to be more or less obstructed, and even obliterated, when aneurisms involve the arch of the aorta.

The formation of an aortic aneurism involves a pre-existing morbid condition of the arterial coats, and in most cases this morbid condition is the atheromatous degeneration. The middle and lining coats becoming softened, distensible, and sometimes destroyed over a certain space, dilatation is produced by the force of the blood-current, and, as a rule, the yielding parts are more and more dilated by the same force. Various circumstances, which are sufficiently obvious, on the one hand favor, and on the other hand retard, the progressive increase of the aneurismal tumor. Other things being equal, the increase in size goes on with a rapidity proportionate to the softened, relaxed state of the sac, the freedom of communication with the artery, the power of the heart's action, and the deficiency of layers of fibrinous deposit. These circumstances varying in different cases, the progress of aneurisms is sometimes extremely slow, and in other cases comparatively rapid.

The primary causes of aneurism affecting the aorta thus are those involved in the production of disease of the coats of this vessel. The supposed agency of muscular exertions or strains in certain cases, irrespective of disease of the artery, may fairly be doubted; but in connection with an atheromatous affection of the arterial coats, they doubtless exert a causative influence. An influence apparently belongs to age and sex. Males are far more subject to the affection than females, and it is rare that it occurs prior to the age of thirty or after the age of sixty. These facts are explained by the more frequent occurrence of disease of the coats of the artery in males than in females, by the infrequency of its occurrence prior to the age of thirty, and by the rigidity of the arterial walls and lessened power of the heart after sixty. The fact that the ascending aorta and the arch are especially apt to be the seat of atheromatous disease, explains, in a great measure, the liability of these portions of the aorta to become affected with aneurism. Aneurisms seated in the smaller arterial trunks are frequently of traumatic origin, but it is obvious that aneurisms of the aorta are never attributable to wounds of this vessel. It is possible that hypertrophy of the left ventricle may contribute to the formation of aneurism, in consequence of the abnormal force with which the blood is driven into the artery.

The terminations of thoracic aneurisms may be stated, before entering on the consideration of the symptoms, signs, and diagnosis. Eventually, in a large proportion of cases, the aneurismal sac opens, and the patient dies from hemorrhage. But in a certain proportion of cases, pressure on important parts, namely, the trachea, bronchi, lungs, œsophagus, spinal cord, the recurrent laryngeal nerve, vena cava, and pulmonary artery, occasions death before rupture takes place. It is needless to say that the existence of aneurism does not preclude the development of various intercurrent affections which may destroy life. The rupture of the aneurismal sac takes place in different situations. It occurs within the pericardium, as already mentioned, when the aneurism is seated below the attachment of this membrane. It also occurs in this situation, occasionally, when the site of the aneurism is above the attachment of the membrane. The latter occurred in five of seventy-nine cases analyzed by Crisp and Swett. The relative frequency of rupture in other situations will be most readily represented by giving the combined results of the statistics furnished by the authors just named. Rupture into the *cavities of the heart* took place in nine of one hundred and thirty-eight cases. Of these nine cases, the rupture was into the right auricle in four, the right ventricle in four, and the left ventricle in one. Rupture into the *pulmonary artery* took place in six of two hundred and seventeen cases. In all of these six cases, the aneurism was seated in either the ascending or transverse aorta. Rupture into the *vena cava* occurred also in six of two hundred and seventeen cases. Rupture into the *pleural sac* took place in fourteen of two hundred and seventeen cases. Aneurisms of the descending aorta are more likely to open in this situation than those seated in the ascending or transverse aorta. The rupture is oftener into the left than into the right pleural cavity. Rupture into the *lung* occurred in eleven of two hundred and fifty-four cases. Rupture into the *œsophagus* took place in sixteen of two hundred and sixty-two cases. When it occurs in this situation the aneurism is generally seated in either the transverse or descending aorta. Rupture into the œsophagus occurred in one of twenty-nine cases which I have recorded since the publication of the first edition of this work, the stomach in this case being filled with blood, but no vomiting of blood taking place. Rupture into the *trachea* took place in thirteen of two hundred and fifty-four cases. The aneurisms in these cases were generally seated at the transverse aorta. Rupture into a *bronchus* took place in eight of two hundred and fifty-four cases. It occurred oftener in the left than in the right bronchus. Of rupture into the *vertebral canal*, only a single instance is contained among the cases analyzed. Rupture *externally* took place in only eight of two hundred and sixty-two cases. It is thus seen that the cases in which the opening takes place in some internal part greatly preponderate over those in which the rupture is external; of two hundred and sixty-two cases analyzed, internal rupture took place in one hundred and forty-five. As already stated, rupture is a termination in a large proportion of cases; but the number of cases in which death occurs either from results of the aneurism irrespective of rupture, or from intercurrent affections, is nevertheless considerable. Of two hundred and fifty fatal cases, in ninety-two rupture did not take place. Finally, a spontaneous cure of thoracic aneurism is possible. It can take place in but one way, which has been already mentioned, namely, the obliteration of the aneurismal cavity by means of the deposit of fibrin. After a spontaneous cure has taken place, the sac, filled with fibrin, and remaining attached to the artery, presents the appearance of an extraneous tumor. Obsolete aneurisms were regarded as tumors formed without the artery, by Corvisart and others, prior to the researches of Hodgson. They undergo more or less reduction in size in the progress of time, from contraction of the contained fibrin, and cases are reported in which the tumor entirely disappeared, leaving only a callosity of the artery where the aneurism had existed.

When rupture of an aneurism occurs, the character of the opening, if it takes place into a serous cavity, differs from that upon a mucous surface or the skin. The serous membrane gives way, or is torn by the force of the current of blood, that is, the opening is a true rupture; whereas, on the mucous surface or skin the opening is by a process of ulceration or a small eschar. Not infrequently several minute openings occur on a mucous surface and the skin. The difference just stated accounts for the fact, that the patient almost always dies quickly from hemorrhage when the opening is into a serous cavity; whereas, death is sometimes preceded by repeated hemorrhages when the opening is upon a mucous surface or the skin. Cases have been reported in which an opening on a mucous surface was quickly closed by a clot, the hemorrhage being thus arrested, and healing of the opening took place. In the case of the celebrated surgeon, Liston, a hemorrhage occurred from an opening of the aneurism into the trachea five months before his sudden death from a second opening in the same direction, there having been, in the meantime, an almost complete immunity from symptoms of the affection. Dr. Gairdner reports a case under his own observation, in which an opening into the trachea took place, which healed, and the patient lived for four years afterward.[1]

SYMPTOMS OF THORACIC ANEURISM.

The symptoms of thoracic aneurism are mainly due to pressure of the aneurismal tumor on the surrounding parts. If the tumor be small, and so situated as not to contract adhesions with, nor press upon, certain portions of the intra-thoracic organs, it may remain latent for an indefinite period. Rupture and sudden death occur not very infrequently when there had been no symptoms to excite suspicion of the existence of aneurism. Aneurismal tumors, however, generally, after they have attained to a certain size, give rise to symptoms more or less marked and characteristic, referable to the respiratory system and voice, the function of deglutition, the venous circulation, and the arterial pulse.

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[1] Clinical Medicine, Edinburgh, 1862. In a case under my observation since the revision of this work, a profuse hemorrhage into the trachea from an aneurismal sac took place three months before the occurrence of a second and fatal hemorrhage in the same situation, the patient in the interim being up and about, and travelling repeatedly by railroad to and from the city of New York, a distance of over fifty miles.—*Note to second edition.*

If the tumor press on either the trachea, or a bronchus, so as to diminish considerably the calibre of these tubes, or if it encroach largely on the space which the lungs should occupy, embarrassment of respiration occurs, manifested especially when an unusual demand is made on the respiratory function as in active muscular exercise. The enlargement of the tumor, however, being gradual, the diminished calibre of the tubes, or the displacement of the lung-substance, although considerable, does not uniformly occasion notable want of breath even on exercise. But in some cases dyspnœa is a very prominent symptom. Pressure on the trachea sufficient to occasion dyspnœa renders the respirations slow and labored, the soft parts above the clavicles being depressed, and the lower part of the chest contracted in the act of inspiration, as in cases of laryngeal or tracheal obstruction from other causes. Aneurisms seated at the upper or posterior aspect of the transverse aorta are those most likely to press upon the trachea. Obstruction of one of the primary bronchi occasions want of breath on exercise, but dyspnœa is generally not experienced when the patient is quiet. The left bronchus is oftener obstructed than the right. The dyspnœa from tracheal obstruction varies at different times, owing to difference in pressure according to the varying size of the aneurismal sac from distension with blood. In certain cases partial relief is obtained when the patient assumes a position in which the aneurismal tumor gravitates from the trachea. Thus in one case under my observation the labor of breathing was diminished by bending the body forward with the arms resting on the knees. In another case the patient habitually inclined the body to the right side, when either sitting or lying, finding in this posture comparative ease. Considerable obstruction by pressure on the trachea occasions a whistling sound with respiration and cough, audible at a distance from the patient. This sign, called stridor, will be noticed presently in connection with the diagnosis of aneurisms.

The aneurismal tumor may extend into the thoracic space, the tumor, in proportion to its size, compressing and displacing the substance of the lungs. This is another source of embarrassment of respiration. Habitual or constant dyspnœa is not usually present under these circumstances, but the patient experiences want of breath on exercise in proportion to the amount of encroachment on the lungs by the aneurismal tumor.

Pressure upon, and irritation of, the recurrent laryngeal nerve are other causes of embarrassment of respiration. From the anatomical relations of the left recurrent laryngeal nerve to the aorta, this, much oftener than the nerve on the right side, is liable to become compressed and irritated; and it is involved, especially in aneurisms seated at the left portion of the transverse, or the upper part of the descending aorta. Aneurisms, however, seated at the right portion of the transverse aorta, or the upper part of the ascending aorta, sometimes extend in a direction to cause pressure or irritation of the right recurrent laryngeal nerve; and sometimes both the right and the left nerve are involved. Respiration is affected through this nerve in two ways: *First*, by pressure on the nerve the respiratory movements of the glottis may be arrested on one side, and occasionally on both sides, more or less obstruction at the glottis being produced, together with laryngeal stridor. *Second*, irritation of the nerve causes spasm of the glottis. The spasm may occasion great obstruction, occurring in paroxysms, and attended by a sonorous or croup-like stridor. Paroxysmal dyspnœa thus attributable to irritation of the recurrent laryngeal nerve, is sometimes extremely distressing, and death from suffocation caused by spasm of the glottis has occurred in several cases under my observation. In one case which I reported for the *American Medical Times* (1864), the patient dying in an attack of spasm, frequently recurring severe paroxysms having induced great exhaustion, there was found after death a calcareous mass between the left recurrent laryngeal nerve and the aneurismal sac, so situated that it was evident irritation of the nerve must have been produced by the expansion of the aneurismal tumor from the blood propelled into it. In this case the aneurism would probably not have proved fatal for a considerable period, had not laryngeal spasm been produced, and the conviction after the autopsy was that the patient might have been spared much suffering and his life prolonged by the introduction of a tube into the trachea.

In one of the cases characterized by the occurrence of spasm of the glottis, tracheotomy was resorted to, to avert impending death from suffocation. The patient was admitted into Bellevue Hospital at evening, and during the night he suffered greatly from dyspnœa. On the following day the respiration was somewhat labored, but he was comparatively comfortable. The dyspnœa returned at night, and when the trachea was opened he was apparently moribund. After the tube was inserted, the breathing was much less labored, consciousness returned, and the pulse became developed; but the relief was only partial, showing that all the obstruction was not at the larynx. Death took place on the following day. The autopsy revealed an aneurism at the transverse aorta of the size of an orange, pressing upon the trachea, and involving the innominate artery together with the right recurrent laryngeal nerve.

It is to be added to the foregoing account of symptoms referable to the recurrent laryngeal nerve, that Todd and Gairdner have observed fatty degeneration and atrophy of the muscles of the side of the larynx corresponding to the nerve compressed by an aneurismal tumor.

Dyspnœa, not due to laryngeal or tracheal obstruction, nor to encroachment by the aneurismal tumor on the thoracic space, nor to pressure upon a bronchus, occurs frequently in some cases of aneurism, in conjunction with the signs of spasmodic or true asthma, namely, labored expiration, dry bronchial rales, vesiculo-tympanitic resonance on percussion, and enlargement of the upper and middle thirds of the chest anteriorly. Spasm of the bronchial muscular fibres in these cases is attributed to pressure of the aneurism on the branches of the pulmonary plexus of nerves. As a symptomatic event in cases of aneurism this is rare.

Impairment of the voice and aphonia are symptoms denoting paralysis affecting the recurrent nerve, which, in some cases, are highly significant. They are apt to occur especially when the aneurism springs from the left side of the transverse portion of the arch. Dependent on the situation of the tumor being such as to occasion pressure on the recurrent nerve, these symptoms characterize in a striking manner certain cases, whereas in other cases the voice remains unaltered, notwithstanding the occurrence of dyspnœa and stridor from laryngeal spasm. Hoarseness, feebleness, or extinction of the voice, if aneurism be not suspected, may lead the practitioner to infer the existence of laryngitis. Tracheotomy has been repeatedly performed under this inference. Stokes has indicated a point of distinction between functional affection of the voice arising from pressure on the recurrent nerve, and the alteration dependent on laryngeal disease, namely, in the latter the hoarseness or aphonia is constant and uniform, whereas in the former remarkable variations in the tone and power of the voice frequently occur within short spaces of time. In a case of aneurism of the innominata, in which the recurrent nerve was found stretched over the tumor like a broad ribbon, the variations of voice were truly remarkable. "Within twenty-four hours it would change from the highest treble to a deep bass; at one time it was an inaudible whisper, at another, hoarse and croaking; and this variability continued up to the period of death."[1] The hoarseness or aphonia due to aneurism may disappear for a time, and again return. This occurred in a case under my observation. In this case the alteration of the voice was the first symptom which indicated the existence of any disease. The patient supposed that he had taken cold, and came to the hospital to be treated for an affection of the air-passages. Up to that time he had felt no inconvenience in performing active manual labor. Bulging at the top of the sternum, and abundant evidence of an aneurismal tumor pressing on the left bronchus and suppressing respiration in the left lung, were apparent on an examination made some weeks afterwards. In cases of aphonia dependent on paralysis affecting the recurrent nerve or nerves, the laryngoscope shows immobility of the vocal chords, together with the absence of appearances denoting inflammation or lesions within the larynx.

Cough and expectoration are incident to inflammation of the trachea excited by pressure of the aneurismal tumor, and to bronchitis produced by pressure on a bronchus or on the substance of the lungs. Cough, without expectoration, may be due to irritation of the pneumogastric or the recurrent laryngeal nerve. In the latter case, the cough may be spasmodic and croup-like in character. Bronchitis may, of course, occur in cases of aneurism, without any pathological connection with the latter; and if there be much obstruction of the trachea from pressure, the difficulty of expectoration renders the coexisting bronchitis a troublesome, and it may be, a serious affection. The accumulation of mucus in the bronchial tubes, under these circumstances, is sometimes the immediate cause of a fatal termination.

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[1] Op. cit., p. 585.

Pressure of the tumor on the œsophagus interferes with the function of deglutition, giving rise to dysphagia from obstruction. This is liable to occur when the site of the aneurism is at the transverse or descending aorta. It is far less frequent than the occurrence of symptoms referable to the respiratory system. It may coexist with the latter, but is sometimes present without them. It may be the only prominent symptom, and, if aneurism be not suspected, stricture of the œsophagus will then be likely to be inferred. As a judicious precaution, an examination should be made for the signs of aneurism in cases of dysphagia dependent on obstruction seated below the pharynx, before resorting to the use of the probang, since rupture of the aneurismal sac has been produced by the passage of this instrument. The difficulty of deglutition varies, of course, according to the amount of obstruction; it may be slight, or the ingestion of solid food may be impossible so that the body suffers from inanition. When the obstruction is extreme, the attempt to swallow, especially solids, frequently provokes paroxysms of pain and spasm, together with cough and dyspnœa, followed by regurgitation of the food arrested in its progress down the œsophagus. The patient refers the seat of the difficulty to the top or middle of the sternum, and sometimes to the epigastrium. Stokes cites a case reported by Law, in which the patient could not swallow in the recumbent position, but always took his food while sitting up, with the body bent forward and to one side. A similar case has fallen under my observation. The explanation of this is sufficiently obvious. The dysphagia has been observed to diminish and even disappear as the aneurism increased in size, a fact to be accounted for by supposing that with the lateral extension of the tumor, the direct pressure on the œsophagus was lessened. The degree of difficulty may be pretty uniform, or it may vary much at different times, owing to variation in the amount of distension of the aneurismal sac, or to the development of spasmodic action in addition to the pressure. Functional dysphagia may be produced by pressure of the aneurism on the pneumogastric nerve. The dysphagia, as thus caused, is paroxysmal, or the variations in the amount of difficulty at different times are much greater than when it is due to the pressure of the aneurismal tumor on the œsophagus. In a case observed by my colleague, Prof. McCready, frequent vomiting and pyrosis seemed to be fairly attributable to irritation of this nerve.

Pressure on the superior vena cava, or the venæ innominatæ, gives rise to venous congestion of the face, neck, and upper extremities. The veins of the neck on one or both sides are distended and tortuous, giving rise, in some cases, to a varicose appearance. The face may be congested to such an extent that it presents a deeply livid and swollen aspect. The neck is sometimes puffed out by vascular turgescence and œdema, forming what Stokes calls a "tippet-like swelling." The distension of the veins, the lividity and œdema, may extend to one or both of the upper extremities. Venous congestion and œdema, thus limited, point to obstruction seated, not at the centre of the circulation, but in the venous trunks which have been mentioned. These veins are likely to become involved in aneurisms springing from the ascending and transverse aorta. Absence of jugular pulsation is a point distinguishing the congestion due to obstruction seated above the heart, from that arising from cardiac lesions which involve tricuspid regurgitation. Physical exploration, perhaps, shows that, in connection with notable congestion apparent only above the heart, cardiac disease is either slight or wanting, and thus affords additional evidence of the seat of the obstruction. Under these circumstances, the existence of an aneurismal or other intra-thoracic tumor pressing on the veins which return the blood from the head and upper extremities is almost certain. Venous congestion, as just described, is present in only a certain proportion of the cases of aneurism. Like the other symptoms, its absence is not proof that aneurism does not exist; but when marked in the upper portion of the body and wanting below, in conjunction with other symptoms, and with signs pointing to aneurism, it is highly significant.

Inequality, as regards size and force, of the radial pulse on the two sides, and the loss of the pulse on one side, are effects of the obstruction of the arteria innominata or the left subclavian incident to certain cases of aortic aneurism. These effects become important symptoms taken in connection with other symptomatic phenomena. The pulse on the left oftener than on the right side is weakened or suppressed, the left subclavian from its situation being the most exposed to pressure. In comparing the pulse on the two sides, it is to be borne in mind that it is normally somewhat more developed in the right than in the left arm; relative weakness on the right side is therefore more significant of disease. The carotid as well as the subclavian artery on one side may be obstructed so that pulsation in this artery and its branches is relatively feeble or extinct. These effects on the arterial pulse, in general, denote that the aneurismal tumor springs from the arch of the aorta. Owing to a change in the direction of the tumor, arterial pulsation, which had been at one time suppressed in the neck or wrist, may be subsequently restored; and, for the same reason, having been weakened, it may become stronger. In these cases, the weakness and suppression were due to the outward pressure of the aneurismal tumor on the subclavian, carotid, or innominata; but in the cases in which these arterial trunks are obstructed from within by fibrinous deposit, the deficiency or absence of pulsation is likely to remain unaltered.

The differences between the radial pulses, due to pressure upon the arteries just named, or to thrombosis, are manifested to the eye by means of the sphygmograph. Retardation of the pulse on one side, as compared with the other side, is a point of difference in some cases in which disparity in other respects is not marked. The pulse in one wrist occurs distinctly after the pulse in the other wrist. This is due to the delay caused by the passage of blood into and from the aneurismal sac. Compression of an aneurismal tumor is found generally to increase the arterial tension as shown by a diminution of the line of ascent delineated by the sphygmograph. By comparing the graphical representation of the pulse, with and without compression, oscillations are observed which, according to Marey, are highly diagnostic of aneurism.

The symptoms which have been noticed are important in aiding to determine the existence of an aneurism and its probable seat. These symptoms may be present, individually or collectively, in different cases; each may exist without the others, and all may be wanting. Singly or combined, they are not pathognomonic of aneurism. Being mostly the immediate effects of eccentric pressure, they may be alike produced by any intrathoracic tumor. Hence their diagnostic value depends on other evidence of the existence of aneurism being conjoined, especially that furnished by physical signs to be presently considered. Other symptoms less characteristic and consisting of secondary or remote effects remain to be briefly noticed.

More or less pain usually attends the progress of thoracic aneurisms. Pain, however, is less constant and less marked as a symptom in aneurisms seated within the chest than within the abdomen. Aneurism of the abdominal aorta frequently gives rise to intense, persisting pain, while this is true of only a small proportion of cases of aneurism affecting the thoracic aorta. Of thoracic aneurisms, those springing from the descending aorta are far more apt to give rise to pain than those seated in the ascending aorta or at the arch. The pain is especially marked if the aneurismal sac cause erosion of the bodies of the vertebræ. In these cases, patients describe the pain as boring or gnawing in character, and it is sometimes referred to a small circumscribed portion of the vertebral column. Aneurisms seated in the ascending or transverse aorta are often unattended by severe pain, but in some cases it is a prominent symptom. In these cases, it is generally intermittent, and resembles that of a neuralgic affection, being lancinating in character, shifting its situation, and shooting in various directions. In other cases, an obtuse, persisting pain is complained of. The pain may be referred to different portions of the chest, extending not infrequently to the shoulders, neck, and arms. The neuralgic pain, in some cases, occurs in paroxysms having all the characters of angina pectoris well marked; this affection, which has been considered in Chapter VI of this work, in a certain proportion of cases, is incidental to aortic aneurism. When an external tumor makes its appearance, pain may be referred to the part where it appears; and prior to this event, pain in some cases is not present. The parts over an aneurismal tumor are often tender, rendering pressure and percussion painful.

Paraplegia becomes a symptom of aneurism, if, when seated in the descending aorta, it leads to erosion of the vertebræ, and the pressure of the tumor falls upon the spinal cord.

Hemiplegia may occur as a remote effect. Clinical observation shows its occurrence in a certain proportion of cases; but it may be dependent on disease of the cerebral arteries, analogous to that which, in the aorta, preceded the formation of aneurism. Under these circumstances, its occurrence may be merely due to coincidence. Exclusive of these instances, it is sometimes due to a fibrinous mass detached from within the aneurismal sac and arrested in one of the arteries of the brain, that is, to embolism. Stokes considers that it may be due to diminished supply of blood in the cases in which the carotid artery on one side is obstructed. Cerebral symptoms, on the other hand, are attributable to congestion arising from pressure of the aneurismal tumor on the vena cava. In connection with the appearances denoting interruption of the return of blood to the heart, drowsiness, dulness of the intellect, obtuse pain in the head, and other signs denoting passive congestion of the brain, are usually present. It is intelligible that the vascular fulness in these cases should favor the occurrence of extravasation giving rise to apoplexy and hemiplegia.

A case was reported, with the morbid specimen, at a meeting of the New York Pathological Society, by Dr. Stephen Rogers, in which the fatal result seemed fairly attributable to cerebral anæmia. In this case the arteria innominata and the left carotid artery were completely occluded by pressure of the aneurismal sac, the left vertebral artery being the only vessel remaining for the supply of blood to the head. For four weeks before death the patient fell into syncope on the slightest exertion, and death at length took place ten hours after a fit of syncope from which the patient did not rally. There was no rupture of the aneurism, and no apparent cause of death except the syncope from cerebral anæmia.[1] If an aneurismal sac attain to a great size it is intelligible that a sufficient quantity of blood may be withdrawn from the circulation to induce general anæmia.

The existence of aneurism does not necessarily involve, directly and speedily, any notable change in the general aspect of the patient. Patients sometimes preserve their weight and strength to the last, but in other cases both undergo more or less diminution. From an analysis of seventeen cases of aneurism seated at the arch, Walshe is led to the conclusion that the difference in different cases as regards loss of weight and strength, is mainly owing to the presence or absence of severe pain. In proportion as this element is prominent, patients emaciate and become enfeebled. Extreme emaciation is sometimes produced by pressure of the aneurismal tumor on the thoracic duct. In some cases there are no symptoms leading the patient to suspect any trouble, prior to the appearance of a pulsating tumor. One of the cases which I have recorded afforded a striking illustration of this fact. The patient, a quartermaster in the army, while travelling in Texas, noticed a pulsation between the first and second ribs on the right of the sternum. Up to this time and afterward he considered himself well, continuing to perform active service, not thinking that the pulsation denoted anything of importance. He subsequently died from rupture of the aneurism. Notable relief from pain, dyspnœa, and other symptoms referable to pressure of the aneurismal tumor on parts within the chest, is sometimes experienced after the tumor has made its way through the thoracic parietes. In one of my cases in which the aneurism attained to an enormous size, instantaneous and permanent relief from dyspnœa followed violent muscular exertions in ploughing.

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[1] *Vide* New York Medical Record, January 15th, 1869.

Dr. W. T. Gairdner was led to observe contraction of the pupil on one side, as a symptom of aneurism, in 1855. As a symptom of an intra-thoracic malignant tumor, it had been previously observed by Dr. MacDonnell, of Montreal. Subsequently to the report of a case by Dr. Gairdner, it has been found by other observers that this symptom occurs in a certain proportion of cases. It was noted in 2 of the 29 cases which I have recorded since the publication of the first edition of this work. It is probable that dilatation of the pupil on one side is also an occasional symptom. The contraction of one of the pupils is explained by the pressure of the aneurismal tumor, at the root of the neck, on the sympathetic nerve. As is well known, section of the cervical sympathetic nerve, in experiments on inferior animals, occasions contraction of the pupil on the corresponding side, an effect attributed to paralysis of the radiating fibres of the iris. Pressure of an aneurismal tumor has a similar effect. This symptom is liable to occur when the aneurism is seated at the upper and back part of the arch of the aorta, "the sac projecting backwards in the direction of the sympathetic trunk, or of its ganglia, and of their communications with the spinal system." It occurs oftener in connection with aneurism of the primary branches of the aorta. Occasionally dilatation of the pupil may be produced by irritation of the nerve or ganglia, an abnormal contraction, instead of paralysis, of the radiating fibres of the iris being thereby produced. Walshe states that contraction and dilatation may alternate in the same case. When the pupil is contracted, it is found to dilate but little if the stimulus of light be withdrawn, and the application of atropia occasions much less dilatation than on the opposite side.

An aneurismal tumor, of sufficient size, may be so situated as to make pressure on the sympathetic nerve of each side and produce contraction of both pupils. In determining the presence of contraction of the pupil, as a symptom of aneurism, it is to be borne in mind, that a slight difference in size between the two pupils is a peculiarity natural to some persons.[1]

Gangrene of the lung is a rare symptomatic event, which, according to Carswell, is induced by compression of the nutrient arteries of the lung by the aneurismal tumor.[2]

PHYSICAL SIGNS OF THORACIC ANEURISM.

The physical signs of thoracic aneurism are furnished by Inspection, Palpation, Percussion, and Auscultation. I shall consider the signs obtained by these methods, severally, following the order in which they are enumerated.

Inspection is frequently not available until the aneurismal sac presses upon the parietes of the chest at some point and gives rise to visible bulging of the surface. The presence of an external swelling or tumor is determined by the eye. At first slight, and limited to a circumscribed area, the swelling may increase so as to form a tumor as large as the fœtal head. The form is usually conical, and the surface, if the tumor be of considerable size, is smooth, and frequently presents a glazed appearance. Pulsatory movements at the site of the bulging, or tumor, may, or may not, be apparent on inspection. A pulsation is sometimes seen when bulging has not occurred. The appearance is then, as remarked by Stokes, as if two hearts were beating in the chest in different situations. This pulsation without swelling may sometimes be discovered by looking across the surface with the eye brought down to a level of the chest, when it is not apparent if the ordinary mode of inspection be alone employed. This is a practical point to be borne in mind.

Palpation enables the observer to ascertain the form of the swelling, the condition of the surface as regards smoothness, and the amount of resistance to pressure. By manipulations with the hand, also, perforation of the thoracic walls may be ascertained; the edges of the eroded ribs or sternum at the aperture through which the aneurismal sac protrudes, may be felt. Fluidity of the contents of the tumor is sometimes apparent to the touch. The liquid contents of the sac may be diminished by manual pressure, and reduction of the hernia-like protrusion perhaps effected. Much compression of the tumor, however, is not to be advised, since there may be risk of producing rupture, or of detaching coagulated fibrin from within the sac, and thus giving rise to emboli, as well as weakening the aneurismal walls.

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[1] With reference to this symptom, see Dr. Gairdner's work on Clinical Medicine; also, a paper by Dr. John W. Ogle, in Medico-Chirurgical Transactions, London, vol. xii, 1858.

[2] Stokes, op. cit., p. 587.

Palpation is especially useful in determining the presence and character of pulsatory movements. Assuming that bulging exists, pulsation constitutes important evidence of its being aneurismal. As a rule, an external aneurismal tumor is pulsatile; but to this rule there are exceptions. If the sac be nearly or quite filled with solid fibrin, the stream of blood through the artery small, and the heart's action weak, an impulse, visible or tactile, may be wanting. On the other hand, intra-thoracic tumors not aneurismal, often present distinct and strong pulsation. The impulse varies greatly in strength in different cases, being sometimes extremely powerful, raising with force the head applied for auscultation, and accompanied by a shock which agitates the whole body, and of which the patient is painfully conscious; in other cases it is scarcely perceptible, and between these extremes every degree of gradation may be observed. The character of the aneurismal impulse, when strong, as Stokes justly remarks, differs from that of the heart's beat in the state of health or of active hypertrophy. The difference arises from the fact that in aneurism the impulse is due to the momentum communicated to a column of liquid, while the beat of the heart is owing to the pressure of the apex of the organ against the chest in its elongating and rotating movements. Quoting the language of the author just named: "The aneurismal beat generally gives the idea of a forcible blow, having a force equal in all directions, while that of the heart conveys the sensation of a mobile but solid body, which, in many instances at least, presents its greatest force at a particular point."[1] This character of impulse, and its strength, are proportionate to the proximity of the sac to the integument; the deficiency of solid fibrin, or, in other words, the relative amount of liquid blood within the aneurismal cavity; the freedom of communication between the aneurismal cavity and the artery, and the power of the heart's action. The impulse may be single or double. When single, it is synchronous with the ventricular systole, being due directly to the contraction of the left ventricle. If double, the second impulse must be produced by the recoil of the arterial coats following distension of the artery by the onward current of blood; it is thus indirectly due to the contraction of the left ventricle, and coincides in time with the second sound of the heart, or, in other words, it is diastolic. A double pulsation, therefore, consists of a systolic and a diastolic impulse.

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[1] Op. cit., p. 554.

In these remarks it is assumed that the aneurismal sac has led to bulging or an external tumor. But, impulse may be perceived by the touch, as well as by the eye, before bulging is apparent. A throbbing, synchronous, or nearly so, with the systole of the heart, may be felt, over a circumscribed space, at a point more or less removed from the seat of the apex-beat. There appear to be two hearts beating within the chest. The presence of the apex-beat at or near its normal situation, is evidence that the abnormal pulsation is not of the heart itself; and that it is not the apex-beat propagated at a distance from the apex, is shown by its being felt within a circumscribed space, and not felt between this space and the point where the apex comes into contact with the parietes of the chest. A source of fallacy connected with the heart may be here mentioned. Free regurgitation through the tricuspid orifice with dilatation of the right auricle and hypertrophy of the right ventricle, may occasion a strong pulsation on the right side of the sternum. An instance related by Stokes has been referred to in a former part of this work.[1] Aneurismal pulsation without bulging, varies in different cases from an intensity exceeding considerably that of the heart's beat, to a feebleness so great that it is almost imperceptible. The throbbing may sometimes be perceived in some cases by placing one hand on the posterior part of the chest, and making firm pressure with the other hand over the upper part of the sternum, when, with a manual examination by one hand alone, it is not appreciable. The pulsation is most evident or marked at the end of an expiratory act. If the aneurism be seated at the transverse aorta and extend upwards, a pulsation may be felt at an early period, before bulging occurs, by passing the finger into the sternal notch, and pressing downward towards the artery. The aneurismal tumor may sometimes be felt in this way, before any visible swelling occurs.

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[1] *Vide* p. 240.

Tactile fremitus, or thrill, with the heart's systole, felt over an aneurismal tumor, is a physical sign which in some cases is strongly marked, but it is by no means uniformly present. It may be present when the impulse is slight or wanting. It is sometimes present in cases of intra-thoracic tumor, not aneurismal. While its absence is not proof that a tumor is not aneurismal, and when present, it does not constitute positive evidence of aneurism, it has, nevertheless, in conjunction with other signs, considerable value, especially if it exist in a notable degree.

Percussion is useful in certain cases by aiding to determine the existence of a tumor, and as affording evidence of its size. If an external tumor have formed, its size, as determined by the eye and touch, is no criterion of the size of the aneurismal sac; the latter extends over an area, greater or less, without the limits of the visible tumor. Its extent may be ascertained with considerable accuracy by means of percussion, provided the pressure on the lung-substance has not induced induration, in which case the percussion-dulness will extend farther than the walls of the sac. It is to be borne in mind that the lung may cover a portion of the aneurismal sac, so that the limits of the latter are not defined by absence of pulmonary resonance. An aneurismal sac, in fact, like the heart, has its area of superficial, and its area of deep dulness on percussion. The former marks the space over which the sac is uncovered of lung; and the latter the distance to which the sac extends beneath the lung. The situation of the dulness goes to show the probable origin of the tumor. Well-defined abnormal dulness over the site of the ascending and transverse aorta, in connection with other signs and with symptoms, points to the existence of aneurism. An aneurismal tumor of small size in the situations just named, if not arising from the posterior margin of the vessel, may occasion an abnormal degree and extent of dulness determinable by careful percussion. Dr. Walshe states that "a sac as large as a good-sized (English) walnut may be discovered, if it lie anywhere between the second and right interspace and the left border of the sternum, and there be no special and unusual source of difficulty in the way." Percussion is less available if the aneurismal sac spring from the descending aorta. When the sac attains to a certain size, however, it gives rise to dulness sufficiently defined behind the interscapular space. It is obvious that percussion, alone, can only furnish evidence that abnormal dulness from some cause exists; that this dulness depends on an aneurism, and not on an intra-thoracic tumor, not aneurismal, or other causes, is to be determined by other signs and by symptoms.

The signs furnished by *auscultation* are referable, not alone to the aneurismal .sac, or the artery with which it communicates, but to the trachea, bronchi, and pulmonary organs. Directing attention first to the signs proceeding directly from the aneurism, it gives rise, in a certain proportion of cases, to a bellows-murmur. This sign is by no means constant; it is not infrequently wanting. Stokes regards it as an accidental sign so commonly wanting that he considers its occurrence as exceptional. Its infrequency is exaggerated in this statement; but, owing to its being so often absent, it has far less value as a sign of thoracic aneurism, than has been generally supposed. As an isolated sign, it possesses very small value for other reasons than its want of constancy. Aortic murmurs are sufficiently common, exclusive of aneurism. It has been already seen that they are often generated by the passage of the blood-current over the internal surface of the vessel, when roughened by atheromatous and calcareous disease, without dilatation. Alone, therefore, a bellows-murmur referable to the aorta is not proof of the existence of aneurism. Other signs and symptoms must be conjoined to render it significant of this affection. It can hardly be said to constitute evidence of aneurism unless the existence of a tumor be determinable, and even then it is not distinctive, since an intra-thoracic tumor, not aneurismal, by pressure on the aorta, may give rise to murmur. The passage of blood into and out of an aneurismal sac gives rise to murmur in a certain proportion of cases. This murmur varies in intensity from the faintest puff to a loudness exceeding that of the most intense cardiac murmurs. It may be soft or rough. It is sometimes most marked over the most prominent portion of the aneurismal tumor, and in other instances it is heard loudest at the base of the tumor. An aneurismal murmur may be single or double.

If single, it is usually, but not invariably, systolic, that is, synchronous with the ventricular systole. The second murmur is synchronous with the second sound of the heart, and may, therefore, be distinguished as diastolic. The latter is rarely, if ever, rough, and is less intense than the systolic murmur.

An aneurismal murmur is to be discriminated from cardiac and arterial murmurs. A murmur emanating from the heart may be propagated to the aneurism. A propagated cardiac murmur may perhaps be more intense over the aneurism, if the tumor be near the surface, than at any point between the aneurism and its source; but its maximum of intensity will be at or just above the base of the heart. If more intense over the aneurism than at or near the base of the heart, it may be considered as not cardiac in its origin. But, in most cases, if a cardiac coexist with an aneurismal murmur, comparison of the two murmurs will show differences as regards quality and pitch, sufficient to denote that they are distinct from each other. A murmur propagated from the heart must be produced at the aortic orifice. A mitral murmur is not conducted along the aorta; hence, when a coexisting cardiac murmur is ascertained to be mitral, its identity with a murmur heard over an aneurismal tumor is not a matter of question. Murmur emanating from the artery, elsewhere than at its point of communication with the aneurism, may be due to disease of the arterial coats, or it may be inorganic, that is, dependent on blood-changes. If due to the former, the maximum of intensity of the murmur will not be likely to be at the aneurismal tumor; if to the latter, auscultation of the neck will show the existence of the venous hum. An anæmic condition doubtless favors the production of an aneurismal murmur and enhances its intensity. Finally, an aneurismal murmur is found to vary at different periods, and even from day to day; and it may exist for a certain period with marked intensity, so as even to be appreciable without auscultation, and subsequently disappear.

Aneurismal sounds, as well as cardiac, are to be distinguished from murmurs. A double sound, corresponding to the systolic and diastolic sound of the heart, is usually heard on auscultation of an aneurismal tumor springing from the aorta. The two sounds resemble those of the heart, not only in rhythm, but in other characters. Stokes remarks: "They are so similar to those of the heart, that, were a good observer blindfolded, and the stethoscope placed for him over the seat of the disease, he would find it difficult, if not impossible, to distinguish them from the ordinary sounds of an excited heart." They are, in fact, the heart-sounds transmitted to the aneurismal sac. That they should be propagated so as to be heard with more intensity over the aneurism than at any point between it and the heart, is readily conceivable in view of the nearness to the ear of the arterial walls and current of blood, when the aneurismal tumor is auscultated. The systolic sound appears to be sometimes reinforced by an element of impulsion derived from the shock communicated to the sac by the onward current of blood; and this element of impulsion is sometimes the only sound appreciable. Either the systolic or diastolic sound may be heard, to the exclusion of the other, and both may be wanting. The latter is likely to occur under the same combination of physical circumstances which renders an impulse extremely feeble or inappreciable.

The auscultatory signs referable to the trachea, bronchi, and pulmonary organs, are due to compression of these parts by the aneurismal sac. These signs may be present before, as well as after, the appearance of an external tumor. They are of considerable importance in determining the existence and seat of aneurism.

Pressure on the lower part of the trachea gives rise, as already stated, to a whistling sound with respiration and cough, which may be perceived at a distance from the patient. If the voice be not affected, it is evident that it proceeds from a point below the larynx. But if there be room for doubt, the stethoscope indicates that it is from below. It may be produced in the bronchus on one side before the aneurismal tumor ascends sufficiently to press upon the trachea. It may be perceived, especially if produced in a bronchus, and referred to its seat, by means of auscultation, when it is not loud enough to be apparent without the aid of the stethoscope. Stokes distinguishes this sign as "*stridor from below.*" It is obvious that it may be produced by a tumor of any kind making pressure on the trachea or bronchi. The practical point is to determine, by auscultation, that a whistling sound, either audible at a distance, or heard with the stethoscope only, is produced, not at the larynx or the upper part of the trachea, but below, at the bifurcation or in a bronchus. If dyspnœa or labored respiration exist, the seat of the obstruction is thus ascertained. The existence of a tumor pressing upon the air-tubes in this situation is rendered highly probable, and it remains for the aneurismal character of the tumor to be shown by other signs.

An aneurismal tumor may compress a bronchus so as to diminish, and even suppress, respiration in one lung. Absence of the respiratory murmur on one side, or a marked disparity in the intensity of the murmur between the two sides, may thus become important signs of the existence and degree of bronchial obstruction. The significance of these signs depends, of course, on the absence of causes of diminished or suppressed respiration on one side, other than occlusion or narrowing of a bronchus by the pressure of a tumor. Bronchitis, emphysema, pleurisy with effusion, and the presence of a foreign body, are to be excluded. That the obstruction is not due to enlarged bronchial glands, or intra-thoracic tumor not aneurismal, is to be determined by other signs pointing to the existence of aneurism.

Finally, absence of the respiratory murmur and of vocal resonance over a circumscribed space, or around an external tumor, concurs with the evidence afforded by percussion, either in rendering probable the existence of a tumor within the chest, not apparent to the eye, or, if visible, in determining the space within the chest which the tumor occupies, to the exclusion of the pulmonary organs.

DIAGNOSIS OF THORACIC ANEURISMS.

A reviewal of the preceding pages will show that the symptomatology of thoracic aneurism furnishes nothing exclusively distinctive of this affection; that is to say, there are no symptoms or signs which are individually pathognomonic. An impulse over a circumscribed space at certain points, distinct from the apex-beat, or a pulsating tumor, renders the presumption strong that aneurism exists; but additional evidence is necessary for a positive diagnosis. Even if bellows-murmur and thrill be added, the existence of aneurism is not unquestionable. The diagnostic force of these signs depends considerably on their degree of prominence. If they be combined and strongly marked, the chances against aneurism are small. If, in addition, the point at which an impulse or a pulsating tumor is observed correspond with the situation in which an aneurism springing from the aorta may be expected to be discovered; and if percussion and auscultation show the presence of an intra-thoracic tumor which can be traced in a direction toward the aorta, there is scarcely room for doubt as to the diagnosis. These diagnostic points are by no means always available. An aneurism may be so situated as regards the thoracic walls, that an impulse is not appreciable, or, if there be a tumor, it may not pulsate, nor present either murmur or thrill. On the other hand, a tumor not aneurismal, may present murmur, pulsation, and thrill. The situation of the tumor is a point of considerable importance. Bearing in mind that, in a great majority of cases, an aneurism springs from the ascending or transverse portion of the aorta, and makes its appearance externally either to the right of the sternum, or at the upper or middle portion of the sternum, or to the left of this bone, a tumor thus situated is likely to prove to be aneurismal. If not aneurismal, it is probably a carcinomatous mediastinal tumor. The latter lying over the aorta may pulsate pretty strongly, and by pressing on the artery may develop a bellows-murmur. Carcinomatous disease, however, in this situation is a much rarer affection than aneurism, so that the chances of the latter preponderate. Age and sex are to be taken into account. A tumor occupying a site in which an aneurism is apt to make its appearance, is more likely to prove to be aneurismal if the patient be a male and between the ages of thirty and sixty. In general, with proper knowledge and care, taking into view all the circumstances of the case, if an aneurismal tumor be apparent externally, its character may be ascertained without great difficulty, and the diagnosis made with positiveness. It is hardly necessary to notice the differential points which distinguish an aneurismal tumor from pulsating empyema, or pericarditis with effusion, for the existence of these affections is readily determined by their proper diagnostic characters.

An aneurism not in contact with the thoracic walls so as to give rise to an impulse appreciable by the eye or touch, and not forming an external tumor, offers a more difficult problem in diagnosis. Certain symptoms which have been considered, namely, embarrassed breathing, stridor, dysphagia, venous congestion of the face and upper extremities, inequality of the pulse on the two sides, functional aphonia, spasm of the glottis, and contraction of one of the pupils, should excite suspicion of aneurism, especially if more or less of them be found in combination, and, also, individually, provided they are not obviously referable to other morbid conditions. Physical exploration, under these circumstances, is essential, not alone in ascertaining signs which point directly to the existence of aneurism, but by showing that other morbid conditions which would account for the symptoms do not exist, and thus leading to a diagnosis, indirectly, by way of exclusion. The chest is to be carefully examined with reference to cardiac disease, since the symptoms and physical signs of aneurism of the ascending and transverse aorta have many points in common with those referable to the heart. The absence of cardiac murmurs and of enlargement of the heart warrants the conclusion that the symptomatic phenomena are not due to lesions of this organ. But even if the heart be not free from disease, it may be sufficiently clear that certain symptoms and signs are not of cardiac origin. Thus, a mitral regurgitant murmur is easily distinguished from a murmur referable to the aorta; and the amount of enlargement of the heart may be obviously inadequate to account for the disturbance of the respiration and circulation. Auscultation of the lungs and air-passages is to be employed in order to ascertain whether the respiratory murmur be diminished or suppressed on one side from compression of a bronchus, and whether the respiratory sound in the larynx and trachea be pure. If stridor be heard, the stethoscope will show the point at which it is produced, and thus indicate the seat of the obstruction. Percussion and auscultation conjoined are to be resorted to in order to ascertain whether there be not dulness, together with absence of the respiratory murmur and vocal resonance, within a circumscribed space so situated that the presence of a tumor having relations to the aorta similar to those which aneurisms are known to have is rendered highly probable or almost certain. A bellows-murmur either localized in this circumscribed space, or heard here with maximum of intensity, and the heart-sounds abnormally transmitted to the same space, are signs entitled to a certain amount of weight. Availing himself of the diagnostic points just recapitulated, the diagnostician may be able to decide on the existence of aneurism, not with absolute certainty, but with much positiveness. The liability to error proceeds from the possibility of enlarged bronchial glands, or intra-thoracic tumor of some kind, not aneurismal, pressing on the trachea, or a bronchus, the subclavian or innominata, the recurrent nerve, the œsophagus, and the vena cava, so as to give rise to more or less of the symptoms and signs due to the pressure of an aneurismal tumor on these parts. The probability of the tumor being aneurismal is considerably increased if the patient be over thirty years of age and of the male sex.

In connection with the subject of diagnosis, it may not be amiss to call attention to the importance of examining for a visible impulse with the eye brought to a level with the surface of the chest; and, for a tactile impulse, with one hand applied to the posterior, and the other to the anterior portion of the chest, making firm pressure with the latter hand. On the very day this paragraph is penned, a case has fallen under my observation in which an impulse was rendered apparent to the eye and touch by these methods of examination only. It is true, that an impulse is not proof positive of the existence of aneurism, but, even if not strongly marked, its value as a diagnostic sign is considerable. It may be important to caution the young observer against mistaking pulsation of the subclavian artery for an aneurismal impulse. It is to be borne in mind that pulsation of this artery is sometimes visible and felt, and especially in cases of disease of the heart involving aortic regurgitation; but, under the latter circumstances, the carotid and other arteries are observed at the same time to pulsate strongly.

Symptoms and signs pointing to the existence of aneurism, are far less available for diagnosis when the aneurismal tumor springs from the descending aorta, than when it is seated at the ascending portion or arch of the vessel. As regards symptoms, labored breathing, stridor, aphonia or hoarseness, venous congestion of the upper portion of the body, and inequality of the pulse, are not produced unless the tumor attains to a great size. Dysphagia from obstruction of the œsophagus is liable to occur, and this symptom, when not connected with disease of the pharynx, should always excite suspicion of aneurism. The frequent occurrence of persisting, boring, or gnawing pain, referred to a particular portion of the spinal column, is to be borne in mind. This should suggest to the mind the possibility of aneurism. The same is to be said of a disposition to keep the body bent forward, in consequence of pain in assuming the erect posture, and of the occurrence of paraplegia, the latter proceeding from erosion of the vertebræ by the pressure of the aneurismal tumor. As regards signs, obstruction of the trachea or a bronchus, and diminished respiratory murmur on one side, are not likely to occur in cases of aneurism seated in the descending aorta. But percussion in the interscapular space may show dulness within a circumscribed area, the limits of which may also be defined by abrupt cessation of respiratory murmur and of vocal resonance. A bellows-murmur may be discovered within this area, not transmitted from the heart, and possibly the heart-sounds may be unduly audible. These signs are not conclusive, but they point to the existence of aneurism. A positive diagnosis is hardly practicable prior to the development of external bulging or a pulsating tumor. So latent are aneurisms in this situation in some cases that, although symptoms denoting some indefinite ailment have been long experienced, an external tumor may be the first event which excites suspicion of the nature of the disease. Physical exploration is of great value in these cases by enabling the diagnostician to exclude certain affections, for example, chronic pleurisy or empyema, for which, otherwise, the disease might be mistaken.

The liability to error in attributing to laryngitis the symptoms caused by the compression or irritation of the recurrent laryngeal nerve in some cases of aneurism has been referred to. Dyspnœa, hoarseness or aphonia, and stridulous breathing, may proceed from paralysis affecting the glottis from compression of this nerve. The laryngoscope should be employed. Ocular inspection, by means of this instrument, will show the existence of paralysis and the absence of inflammation; and whenever the symptoms just named are thus attributable to paralysis, aneurism is to be suspected, and the signs of an aneurismal tumor are to be sought after.

Paroxysmal dyspnœa, with stridor accompanying respiration and cough, is certainly not caused by laryngitis if the voice, in the intervals, be neither raucous, stridulous, nor abolished. And laryngeal spasm, not associated with laryngitis, occurring in a person of middle age, and especially in a male, should always be considered as pointing to the existence of aneurism.

Another liability to error is incident to cases in which an aneurismal tumor, springing from the outer aspect of the ascending aorta, extends into the right side of the chest and encroaches considerably upon the pulmonary space. The physical signs, as well as symptoms, in such a case, may seem to denote tuberculous affection. If none of the characteristic symptoms and signs of aneurism are present, this error in diagnosis is very likely to be made. I can bear testimony to this from my own experience.

Aneurism of the arteria innominata is to be discriminated from aortic aneurism. In the former the sac tends more to extension in an upward direction, the tumor appearing more speedily, and found above or behind the sternal portion of the right clavicle. The clavicle is frequently raised. Pressure on the nerves going to form the brachial plexus is apt to occasion more or less paralysis of the right upper extremity. A differential point stated by Dr. Holland is, that when pressure is made on the subclavian or carotid artery of the right side, the pulsation of the aneurismal sac is suspended if the aneurism be seated in the innominate artery, whereas the pulsation is not affected if the aneurism be aortic.

TREATMENT OF THORACIC ANEURISM.

Recovery from thoracic aneurism is possible. The chances against recovery, however, preponderate so vastly that in any given case there is but little ground to hope for this result. Still, the possibility of recovery is not to be lost sight of in the management of the cases which it is the misfortune of the physician to meet with in practice. But although recovery is not be looked for, the progress of the affection may be more or less slow, and the physician may reasonably hope to contribute, by judicious management, to postpone for an indefinite period the fatal result. Moreover, the palliation of distressing symptoms, always an important object of treatment, furnishes scope, in the progress of this affection, for the useful application of remedies.

Recovery from thoracic aneurism can only be effected by the deposit within the sac of successive layers of solid fibrin until the cavity is obliterated, leaving the channel of the artery free. Can this curative process be promoted by medical treatment? There are no medicines which exert a special effect for this end. The general conditions favorable for the process are, an abundance of the fibrinous constituent of the blood, with an equable and not too active state of the circulation. These are conditions which are to some extent controllable. Those which depend on local circumstances, such as the size and shape of the cavity, the form and direction of the aperture, the state of the interior of the sac as regards roughness, &c., are obviously beyond control. The indications for treatment, then, with a view to the possibility of recovery are to maintain the relative proportion of the fibrinous constituent of the blood, and to secure regularity of the action of the heart, obviating, on the one hand, over-excitement, and, on the other hand, undue feebleness. How are these indications to be fulfilled? In brief, by a nutritious, and, in some instances, a generous diet, embracing a good proportion of animal food; by tonic remedies and stimulants when the appetite and digestive powers are enfeebled; and by the restricted use of liquids, which, by increasing the quantity of blood, lessen the proportion of fibrin to the mass. These measures relate to the first indication, namely, to maintain or increase the fibrin. The second indication, namely, to secure regularity of the heart's action, is to be fulfilled by avoiding active exercise, mental excitement, and other causes inducing disturbance or undue activity of the circulation; by moderate depletion by means of saline laxatives, or resorting perhaps in some cases to small bleedings if decided plethora exist, but on the other hand, prescribing ferruginous remedies if anæmia be present; and by the use of sedatives if the circulation be unduly and persistingly over-excited.

These indications, having reference to the possibility of recovery, relate not less to an object of management more likely to be attained, namely, to retard the progress of the affection, and thus postpone the fatal result. This object is alike secured by the deposit of fibrin within the aneurismal cavity, by which the walls of the sac are strengthened, the tendency to its enlargement being thereby lessened, and by tranquillity of the heart's action. The means, then, by which the physician may hope to prolong life are those which, at the same time, afford the best chance, slight as this is, of recovery.

Potent measures of treatment have been heretofore advised and pursued with the expectation of retarding the progress of thoracic aneurisms and with a faint hope of effecting a cure. The plan of treatment introduced more than a century and a half ago by Albertini and Valsalva, adopted by Laennec, Corvisart, and Bouillaud, and recommended in a modified form by Hope, obtained more favor and currency than any other. This plan consisted in repeated bleedings, confinement to the bed for about forty days, during which period all stimulants were to be abstained from, and the diet reduced to the minimum quantity required for the maintenance of life. After the strength had been so far reduced that the patient was scarcely able to raise the hand from the bed, the quantity of food was gradually increased, and after a certain measure of strength had been regained, absolute rest was no longer insisted on. Cases were reported in which this system of treatment appeared to stay the progress of the aneurism, and in some instances it was stated to have effected recovery. The severity of the system is such that few patients were willing to submit to it and of late it has been for the most part abandoned. With reference to it, as well as to other potent measures, the following remarks by Stokes, are pertinent: "It is to be doubted whether we are ever justified in adopting any measures which, while they are directed, under theoretical views, to the cure of the disease, materially interfere with the patient's condition. It often happens that a patient who has not been thus interfered with will continue with unimpaired health and strength for a great length of time until he is so unfortunate as to be placed under treatment for the cure of his aneurism. For then all the evils which have been pointed out as occurring in cases of indolent disease of the heart, when injured by ignorant treatment, are induced. The patient's mind becomes excited and apprehensive, his system is weakened by depletion, and his digestive functions ruined by starvation. The forces by which he can resist disease are broken down, his blood becomes uncoagulable, his tissues unresisting. The force of the aneurismal throb is augmented, and a disease which, under other circumstances, might have endured for years with but little interference with the general health, is turned into a rapid and destructive malady."[1]

Pursuing no special plan of treatment, the management must depend very much on the circumstances pertaining to individual cases. Is the patient of a full plethoric habit, the vessels over-repleted, and the heart over-stimulated; depletion may be called for, care being taken not to carry it beyond the point of restoring the blood to its normal condition. Is the patient anæmic, and the heart's action excited by impoverished blood; a nutritious animal diet, together with preparations of iron and perhaps porter, wine, or spirits, are important, being careful not to push these measures to an extreme, and thereby induce evils equal to those incident to anæmia. Is the appetite good and digestion active; indulgence at the table is to be restrained. On the other hand is the appetite poor and digestion weak; measures to improve both are indicated. The restricted use of liquids is an important point in the management, conducing, in connection with an appropriate diet, to the formation of blood rich in fibrin, and not excessive in quantity. These ends are promoted by a limited supply of liquid, far better than by the elimination of liquid by means of hydragogue cathartics and diuretics.

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[1] Op. cit., p. 606.

A regular and tranquil condition of the heart being highly desirable in all cases, everything which excites unduly this organ is, of course, as far as possible, to be avoided. Active muscular exercise, mental excitement, the abuse of alcoholic stimulants, &c., are to be interdicted. It is an important question, in this connection, how far exercise can be taken with safety or advantage. Some writers recommend perfect quietude, and enjoining the patient to remain a considerable portion of the time in the recumbent posture.[1] The propriety of this is more than doubtful. The appetite, digestion, and general condition of the body must suffer from such inaction, so that the risk of doing harm by impoverishing the blood and weakening the vital forces, is greater than the liability to injury from moderate exercise. An amount of exercise which can be taken without accelerating the circulation, will be likely to be useful rather than injurious.

An important point in the treatment concerns the mental condition of the patient. A full knowledge of the nature of the affection, and of the accidents to which he is exposed, can hardly fail, in most cases, to induce depression and apprehension, the effect of which must be, to a certain extent, unfavorable. When it can be done without breach of good faith, or a violation of truth, the physician will do wisely in forbearing to enter into an elaborate exposition of the characters, tendencies, and results of thoracic aneurisms. While deception here, as in other forms of disease, is unworthy the character of a physician, as well as unjust to the patient, it is fair to present as favorable a view of the case as facts will warrant. The patient may be encouraged with the hope of the affection progressing very slowly, or remaining stationary for an indefinite period, and even with the possibility of recovery. He will thus be spared not only a portion of the unhappiness to which the affection is calculated to give rise, but the unfavorable influence of excessive anxiety and gloom; and he will, moreover, be more disposed to persevere in following faithfully regulations of diet and regimen.

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[1] *E. g.* Bellingham, op. cit.

If the action of the heart be habitually excited or irregular, sedative remedies are indicated, such as hydrocyanic acid, aconite, hyoscyamus, opium in small doses, &c. It is considered by some writers injudicious to resort to digitalis for this purpose; but I can see no just grounds for apprehending evils from the use of this remedy, if proper caution be observed. Here, as in other instances in which it is desirable to reduce the frequency of the heart's action, it is a remedy of great value.

Certain remedies have been recommended with a view to induce a condition of the blood favoring the coagulation of the fibrin. Laennec, Hope, and others have regarded the acetate of lead as useful in this way. This remedy was employed in one of the cases which I have noted, to the extent of producing the characters in a blue line on the gums, but without any good effect. Walshe attaches value to gallic or tannic acid. Theoretically, remedies are indicated which produce this effect, provided it be not counterbalanced by other consequences. It is, however, doubtful whether these or other known remedies produce this effect; and moreover it is to be considered that ordinary coagulation of blood within the sac, so far from being desirable, would be likely to give rise to serious results. Coagulation is conservative and curative only when it takes place at the bottom of the sac, leading to the formation of layers of fibrin, which become adherent first to the walls of the sac, and successively to each other, until the cavity is more or less filled with the solid deposit.

The various symptomatic and remote effects of thoracic aneurisms, such as pain, cerebral congestion, labored respiration, cough, dysphagia, &c., will, of course, claim palliative measures of treatment, which are to be adapted to the particular circumstances of individual cases, and need not be here considered.

The foregoing remarks on the treatment of thoracic aneurisms, contained in the first edition of this work, are retained without material alteration. Since the publication of that edition, Joliffe Tuffnell, of Dublin, has reported a series of cases which show a remarkable success from a plan of treatment proposed by the late Dr. Bellingham. This plan is known as "Tuffnell's plan." The essential features, however, are contained in the valuable treatise on the diseases of the heart, by Bellingham.

Of Tuffnell's plan, an essential and probably the most important measure is the maintenance of perfect rest in a recumbent posture for two or three months. The patient, during this time, is not to raise himself even to the semi-recumbent posture, but he may turn carefully from side to side, and at times lie on his face. The arrangements should be such that the bowels and bladder may be evacuated without the necessity of the patient being moved in bed. The room should be sunny, if possible, and the situation of the bed such that the patient may be amused by looking out of doors.

The plan embraces a restricted diet, which is to be rigidly adhered to as long as the patient is kept in a state of rest. The rules laid down for diet are precise, and are as follows: For breakfast and supper, two ounces of white bread and butter, with two ounces of milk, cocoa, or tea; and for dinner, three ounces of meat, either broiled or boiled, and three ounces of potatoes or bread, with four ounces of water or claret. This diet gives for each day, of solids ten, and of liquids eight ounces.

The immediate objects of this plan are, *first*, to secure, by rest, a uniform tranquillity of the circulation; and, *second*, to supply aliment sufficient in quantity and kind for the wants of the system and no more. These objects have reference to the only mode in which a cure of internal aneurism can take place, namely, by the aneurismal sac becoming filled with fibrin, deposited in successive firm layers, transforming the sac into a solid tumor which may remain permanently without change, save progressive diminution in size, the arterial canal being unobstructed.

This plan of treatment is a reproduction, in part, of the system of Valsalva and Albertini. Of the latter, perfect rest for six weeks was an essential feature. The abstraction of blood, which formed an important part of the system of Valsalva and Albertini, does not enter into the plan of Bellingham and Tuffnell. The reduction of diet by the latter is not carried as far as by the former; yet, the diet prescribed by Bellingham and Tuffnell is below that required by the body in health, and can be continued only for a limited period. The period during which complete rest in the recumbent posture is maintained is longer in the plan of Bellingham and Tuffnell.

Dr. Bowditch has suggested, as modifications, the occasional abstraction of blood, in certain cases, either by venesection or leeches, and the employment of cardiac sedatives, such as digitalis, veratrum viride, &c. He also suggests that while the diet should be carefully regulated, the quantity should not be reduced below the requirements of the system for nutrition. The last-named modification would divest the plan of one of the difficulties in the way of faithfully carrying it out, namely, the reluctance of patients to submit to the dietetic restriction.[1]

As regards the most important measure, namely, rest in a recumbent posture, the immediate object, as already stated, is to secure a uniform tranquillity of the circulation, this doubtless being a *sine qua non* in any mode of treatment having reference to the deposition of fibrin in the aneurismal sac. The diminished frequency of the heart's action secured by confinement to the recumbent posture, as well as uniformity of its action, is an object of importance. Tuffnell reports in one of his cases a reduction to the extent of 30 beats, namely, from 96, standing, to 66 lying, a reduction amounting to 1800 beats per hour, and 21,600 beats in twelve hours. Bowditch gives the results of his own observations respecting the variations in the pulse in the sitting, standing, and lying posture. Of five persons in his own family, the average frequency in the standing posture was 81.6; in the sitting posture, 74.8, and in the lying posture 70.0. Of 14 hospital surgical cases, the averages were as follows: Standing, 91.42; sitting, 73.85; lying, 68.07. With respect to the relative importance of rest in the treatment, Bowditch remarks as follows: "Comparing this powerful influence on the rapid flow of blood with the little real influence that we can have on the composition of the blood by any diet, and the doubt one has as to whether the increase of fibrin is really a desirable object to be obtained, and that it forms but a very small constituent part of the circulating fluid, I think we are led to suspect that, after all, this element of quiet in a horizontal position is the most important agent of all the three originally proposed even in Hippocratic days, hinted at and followed by Valsalva, and distinctly pressed upon our notice by Dr. Tuffnell, but not so much relied upon as it ought to have been by him or by his predecessors.

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[1] *Vide* paper by Dr. Bowditch, read before the Boston Society for Medical Observation, February, 1866, and communicated for the Boston Med. and Surg. Journal.

Tuffnell has reported 6 cases in which his plan of treatment was carried out. The following is a brief analysis of these cases as given in the paper by Bowditch:

1. An Irish carman, æt. 35; 1851, in hospital; aneurism, abdominal, aortic, size of an orange. Treatment, horizontal position, three months. Food, eight ounces solids, six ounces fluids, in twenty-four hours. Recovery perfect.

2. Seaman, æt. 39; 1854, in hospital; internal aortic aneurism—undoubted. Ten weeks' perfect rest, and restricted diet. Complete recovery.

3. Die cutter, æt. 54; in hospital; aortic aneurism, projecting through sternum; all the severer symptoms; integuments alone covering tumor. In three months, "general health excellent," and all local signs less; tumor had grown firmer. Finally, he resumed work. Varicose external vessels were seen in great numbers three years afterwards—the man still at work.

4. A fat, indolent merchant, with abdominal aneurism five inches in diameter. In three months, tumor quite solid, though pulsating. Thirteen weeks on his back.

5. A gentleman æt. 30; a hunter and high liver; October, 1855. Aneurism at the bifurcation of the aorta. In nine weeks, better; in six months no pulsation remained. He then made over-exertion, and needed venesection; again, relief. Subsequently, other surgeons saw him, laughed at the idea of there being aneurism, and advised hunting, &c., as before. In eighteen months the aneurism suddenly burst.

6. Laborer, æt. 30; in hospital, January, 1864; abdominal aneurism. Pulse, 104 standing, 66 lying. Disease checked by treatment.

After a perusal of the paper, by Bowditch, in the summer of 1866, I resolved to make trial of Tuffnell's plan whenever I should meet with cases in which patients were willing to conform to it.

The two following cases I reported for the New York Medical Gazette, January 4th. 1868. The seat of the aneurism in each of these cases was the abdominal aorta; but the cases are introduced, inasmuch as the principles of treatment are alike applicable to thoracic aneurisms:

Case 1. Charles Blanson, æt. 32, a native of France, admitted into Bellevue Hospital, Aug. 2, 1866.

The following previous history was obtained by Dr. Forman, senior assistant: "He was perfectly healthy until about eight years ago, when he had an attack of rheumatism. He has since, repeatedly, had attacks of this disease. Four years ago, when serving in the United States army, he was taken ill, complaining of pain in the chest, shortness of breath, and palpitation. The two latter symptoms continuing, he was transferred to the Invalid Corps. In about one year after this attack he had a dull, heavy pain in the back, which has continued, except at intervals, up to the present time. He noticed for the first time, about one year ago, a small pulsating tumor just below the ensiform cartilage. This tumor has gradually increased in size. It did not prevent him from continuing his occupation, that of a waiter, until about five months ago. For the past five months he has been unable to work."

The aneurismal tumor was situated between the median line and the false ribs on the left side. The tumor was about as large as a small orange. The pulsation was strong, lateral as well as anterior, and was accompanied by a loud, systolic, bellows-murmur. There existed in this case an aortic direct and an aortic regurgitant bellows-murmur. The apex-beat of the heart was in the fifth intercostal space, half an inch without the linea mammalis. The superficial arteries pulsated strongly.

The general aspect of the patient was not morbid. The plan of treatment was explained to him, and he was desirous that it should be tried. It was commenced August 14th. On the 27th of August, at the patient's request, corn meal gruel was substituted for bread.

On October 1st, owing to changes of service, the patient came under the care of Prof. A. Clark, who continued the plan of treatment.

Under date of October 21st, I noted that the patient had borne the treatment well, except that, at times, he suffered from diarrhœa, and that pain in the situation of the aneurism was sometimes severe. The tumor, at this date, seemed to have increased in size.

Nov. 11th, 1866. Under this date I made the following note: This patient died ten days ago. After the date of the previous note, *i. e.*, October 21st, he progressively failed. The aneurism evidently increased in size. He suffered from the frequent recurrence of diarrhœa, and died by slow asthenia. The symptoms afforded no evidence of rupture of the aneurism. An autopsy could not be obtained.

This case was made the subject of a clinical lecture at Bellevue Hospital, September 14th, 1866. The patient entered very cordially into the measures of treatment, and I believe they were faithfully carried out.

Case 2. John Brown, æt. 46, native of Ireland, laborer, admitted into Bellevue Hospital, August 31st, 1867.

He stated that he was healthy up to two years before his admission. He had had chancre fourteen years ago, but no subsequent symptoms denoting syphilis. He had been a hard drinker, but had not drank for about two years. For nearly two years he had been subject to pains in the belly resembling those of colic, and also paroxysms of pain referred to the epigastrium. In June, 1867, he first noticed a pulsating tumor just below the epigastrium. Since that date the paroxysms of epigastric pain had been more severe than previously, and accompanied by disorder of digestion.

On his admission, a pulsating tumor was distinctly felt just below the epigastrium, the pulsation being lateral and anterior. The vertical and the horizontal diameter were about three inches. The tumor was resisting on pressure, and dull on percussion. It was easily felt, and the pulsation was strong. There were no signs of cardiac disease. The radial arteries were rigid. The lungs were healthy. The urine was normal.

September 11th. The treatment, after the plan of Tuffnell, was commenced, the patient consenting to its being faithfully carried out.

September 27th. I noted that the pain in the region of the tumor had diminished, but it was still considerable, and opiates were given *pro re nata*. He was constipated, and a laxative was occasionally prescribed. His general aspect was as good as when the treatment was commenced. The tumor had not increased in size; the pulsation was about the same, but the resistance to the touch seemed somewhat greater. The patient did not complain of pain referable to the back. It should have been stated that, from the first, a systolic and diastolic bellows-murmur were discoverable, over the tumor, with the binaural stethoscope.

Owing to change of service, this patient came under the care of Prof. A. Clark, on October 1st. The plan of treatment was continued by Prof. Clark.

November 4th. I noted that the size of the tumor and the force of the pulsation were evidently diminished. He suffered but little from pain. His general aspect was good.

December 3d. I noted that the tumor did not seem to be more than two inches in its vertical and horizontal diameter, and was notably resisting to pressure. The diastolic murmur had disappeared, but the systolic murmur was still heard. There was no lateral pulsation. The general condition of the patient was good. He complained, however, now of pain in the back. He was very impatient to be permitted to sit up, and to have a more varied diet.

Death occurred suddenly in this case December 8th. The patient became suddenly pallid and faint, and died in about ten minutes. There can hardly be room for doubt that rupture of the aneurism took place. Owing to the refusal of friends in this, as in the other case, an autopsy could not be obtained.

The dietetic part of the treatment in this case, I have reason to believe, was faithfully carried out; but as regards perfect rest in the horizontal posture, the patient was not altogether faithful. On several occasions I discovered him sitting up in bed. Especially during the latter part of the time, he became much dissatisfied, and two days before his death was desirous of leaving the hospital. The case was made the subject of a clinical lecture September 27th, and also December 3d, 1867.

In the first of these two cases the patient was confined to the bed nineteen weeks and two days. In the second case the duration of the confinement to bed was twenty-two weeks. In the first case the plan of treatment did not appear to affect the progressive increase of the aneurism, although the patient did not die from rupture. In this case it is possible that the plan of treatment, as regards diet, was unfavorable, and that the patient might have lived longer had the plan not been pursued. In the second case much hope was entertained that the plan of treatment would succeed, even up to the time of the rupture of the aneurism. The apparent effect upon the aneurismal tumor was all that could have been expected. The occurrence of pain in the back in this case during the latter part of the time the patient was under treatment probably indicated erosion of the spinal column.

Since reporting the foregoing cases, two additional cases have come under my observation, in which Tuffnell's plan of treatment was pursued. A brief report of these two cases is subjoined.

Case 1. Mr. S., accompanied by Dr. William Clarke, of Guelph, Canada, consulted me in June, 1868. Slight hemiplegia had existed for several years. He had aphonia, which a short time before had occurred suddenly. Occasionally he was able to make a loud sound, which was hoarse, not stridulous. The following signs denoted aneurism: evident enlargement on the left of the sternum, just below the clavicle; flatness on percussion within a circumscribed space in this situation, together with a distinct pulsation without thrill, an abnormal transmission of the heart-sounds, and a feeble, low, systolic murmur. The aphonia was attributed to pressure on the recurrent laryngeal nerve. The aneurism pressed on the left primary bronchus, as shown by notable feebleness of the respiratory murmur over the whole of the left side of the chest, the murmur on the right side being notably loud. The left radial pulse was scarcely appreciable, the pulse on the right side being distinct. There were no signs of cardiac disease; the patient had had hæmoptysis.

It was agreed with Dr. Clarke that the patient should return home, and that Tuffnell's plan of treatment should be pursued, the patient consenting to submit to it. The following is an extract from a letter from Dr. Clarke, written seventeen days after the date of the consultation: "After leaving you, I had a sad journey home with Mr. S. The hæmoptysis has recurred twice profusely, and continued slightly up to four days ago. When we arrived at home, I at once confined him to the bed, enjoining strictly the recumbent posture, and he was placed on the allowance of food as we had arranged. He has, in the meantime, considerably improved as regards breathing and voice; but the pulsation of the aneurism extends over a larger space, and is much stronger."

The following is an extract from a letter written by Dr. Clarke forty days after the date of consultation: "Mr. S. continues in nearly the same condition. His strength keeps up on the low diet and a rigid adherence to the recumbent posture. He has had no return of the hæmoptysis. The tumor seems to have enlarged."

Dr. Clarke announced the death of this patient in a letter dated August 29th, as follows: "Our patient, Mr. S., died on the 24th August, after fifty-seven days in the recumbent posture, and under the dietetic treatment which was agreed upon. The diet was always sufficient for his appetite, and for some time before his death he did not eat his allowance. For the last ten days of his life he had paralysis of the bladder and rectum. He did not become greatly emaciated, and his intellect was clear up to the last hour." A report of the autopsy by Dr. H. F. Tuck accompanied this letter. The whole of the transverse aorta was involved in an aneurismal sac measuring nine inches in length and eight inches in diameter. The interior of the sac was about one-half filled with fibrin, the deeper layers being dense, and those which were superficial being of recent deposit. The sac was not ruptured. The heart was somewhat enlarged and flabby. The valves were sound.

CASE 2. Andrew Sheridan, baker, aged 38, was admitted into Charity Hospital, Blackwell's Island, August 26th, 1868. The following notes of the case are furnished by Dr. Dew, House Physician: "The patient first noticed a pulsation a little to the right of the sternum, about five years ago; but at that time he experienced no inconvenience except from slight dyspnœa on exertion. Two years ago he was treated for pneumonia in a hospital at St. Louis, Mo., and the existence of aneurism was then ascertained. On his admission into Charity Hospital, there was a prominence at the median line on the sternum, extending to the right side on a level with the third costal cartilage. The pulsation over this prominence was strong, and there was a loud systolic murmur. He suffered much from dyspnœa on any exertion. Pain in the site of the aneurism was severe. About September 16th, Tuffnell's plan of treatment was entered upon by direction of Dr. Flint. The patient was placed on an air-bed, and the recumbent position strictly enjoined. The diet consisted of two ounces of bread, with butter, and a cup of coffee, for breakfast; three ounces of meat, with bread and molasses, for dinner; and for supper, two ounces of bread, with butter, and two ounces of milk or tea. Pills of lactucarium and hyoscyamus were given to allay restlessness.

"November 20th. About a month ago, four ounces of wine daily were ordered for this patient. He looks now thinner and paler than on his admission, but his general condition is good. Pain in the tumor is less. He complains of pain in the right shoulder. All who have observed the case think that the pulsation, together with the murmur, has diminished, and that the progress is satisfactory.

"December 15th. The restriction as regards diet was removed on the 1st inst.; but the patient still keeps the recumbent posture. The prominence over the aneurism seems to be diminished, as also the pulsation and murmur. He is anxious to be allowed to sit up."

This patient was transferred to Bellevue Hospital, January 15th, 1860. He kept the recumbent posture up to the time of his being transferred; but he stated that in November he was greatly excited on one occasion, and was obliged to make violent muscular exertions in defending himself from the assault of an insane patient in the ward. He remained in Bellevue Hospital to the time of his death, which took place in the early part of September, 1869. The aneurismal tumor attained to an enormous size, and, for several days before his death, ulceration and sloughing occurred at the point most projecting, and the opening of the sac was hourly expected. Rupture, however, did not take place, the mode of dying being by slow asthenia. An autopsy was not made, the patient having made a request to that effect.

The foregoing four cases are here introduced in view of the importance of accumulating clinical experience respecting a plan of treatment which seems to have been remarkably successful in the hands of Tuffnell. The failure in these cases does not, of course, impair the fact that success from this plan, in a certain proportion of cases, may be expected; and I shall not be discouraged in making further trial of the plan, although, I must confess, with expectations of success somewhat less sanguine than heretofore. It is to be considered that aneurisms are not infrequently associated with grave lesions of the heart, or serious affections of other organs, and that death may be due, measurably or entirely, to these, while the deposit of fibrin within the aneurismal sac may be progressing favorably. It is also to be considered that, especially in hospital practice, perfect confidence cannot be felt that the plan of treatment is fully carried out. The plan requires full co-operation on the part of the patient. Without an entire understanding with respect to this point, it is of no use to enter upon it. My experience at this time would have embraced several more cases if the co-operation of the patients at the outset could have been secured. But after the patient has promised, and the plan has been entered upon, his resolution may give way, and, when unobserved, he will be likely to violate the rules respecting recumbency and diet. I am free to admit that each of my hospital cases are open to suspicion on this score, and in one of the cases it was certain that the rules were not rigidly adhered to. It remains to be determined, by an analysis of a large collection of cases, how much importance belongs to confinement in the recumbent posture, and whether it be better to reduce the diet as practised by Tuffnell, or to allow an ample amount of food as suggested by Bowditch. It also remains to be ascertained whether the plan may not be rendered more efficient by the use of remedies which diminish the frequency of the heart's action.

The propriety of resorting to tracheotomy becomes a question in certain cases in which great suffering and immediate danger to life arise from spasm of the glottis caused by irritation of the recurrent laryngeal nerve. Relief, postponement of a fatal termination, and the possibility of recovery, may be secured by the insertion of a tube in the trachea. These attainable objects afford a sufficient warrant for the operation. It is superfluous to add that, in determining to resort to tracheotomy, the nature and seat of the obstruction should be clearly made out. The operation would be admissible when obstruction at the glottis is produced by paralysis of the laryngeal muscles from pressure of the aneurismal tumor on the laryngeal nerve, were it not that the obstruction, as thus produced, does not give rise to great suffering and danger to life; but it would be a misfortune to open the trachea when the interference with respiration arises either from tracheal obstruction below the point where the operation is made, or from causes relating to the heart or lungs.

INDEX.

A.

Adhesions of pericardium, 334, 336, 414
    effects on the heart and circulation, 415
    how ascertained during life, 420
Albuminuria in dilatation, 93
    in valvular lesions, 189
    in pericarditis, 359
Anæmia in valvular lesions, 192, 250
    in connection with enlargement of thyroid body and prominence of the eyes, 311, 319
    in functional disorder of the heart, 460
Aneurism, cardiac, 115
Aneurisms, thoracic, 497
    varieties of, 497
    anatomical relations of, 500
    formation and causes of, 502
    terminations of, 503
    symptoms of, 505
    physical signs of, 516
    diagnosis of, 523
    treatment of, 528
Angina pectoris, 288
    description of, 289
    pathological character and relations of, 291
    infrequency of, 297
    prognosis, 299
    diagnosis, 301
    treatment, 303
    in cases of fatty degeneration of heart, 102
Aortic lesions, 129
        anatomical characters, 129
        frequency of, 137
        effect on left ventricle, 144
            on heart-sounds, 232
        diagnostic characters of, 237
    valve, water test of sufficiency of, 138
    orifice, normal size of, 139
    regurgitation, 142
    contraction, 142
    direct or systolic murmur, 210
    regurgitant or diastolic murmur, 212
Aorta, diseases of, 487
    inflammation of, 487
Aorta, morbid deposit on lining membrane of, 488
    atheromatous deposit in, 488
    calcareous degeneration in, 488
    dilatation of, 491
    contraction and obliteration of, 492
Apex-beat, normal situation and deviations of, 48
    normal force of, 49
    altered situation in enlargement of heart, 50
    enlarged area of, in enlargement of heart, 51
    increased force of, in hypertrophy, 51
    in dilatation, 85
    in pericarditis, 375, 380
    in pericardial adhesions, 422
    in functional disorder, 57
Aphonia in thoracic aneurisms, 508
Apoplexy in connection with hypertrophy, 36
    in connection with valvular lesions, 181
    pseudo, in fatty degeneration, 100
    pseudo, in valvular lesions, 183
    pulmonary, in valvular lesions, 176
Arcus senilis in fatty degeneration, 101
Arteries, obstruction of, by fibrinous deposits from valves and orifices, 168, 182, 436, 502
Asthma, cardiac, 83, 174
Asystole, 84, 103
Attrition, sounds of, in pericarditis, 364
Atrophy of heart, 91
Atheroma in aorta, 488
Auscultation in hypertrophy, 65
    in dilatation, 85
    in fatty degeneration, 103
    in pericarditis, 364, 379
    in pericardial adhesions, 421
    in endocarditis, 440, 447
    in functional disorder, 468
    in thoracic aneurisms, 520

B.

Bellingham on size of the orifices of the heart, 139
Becquerel on cirrhosis and valvular lesions, 187

Begbie on prominence of eyes, 306
Bizot, measurements of heart, 20
  of orifices, 188
Blisters in pericarditis, 395, 401
Blood-currents, *vide* currents of blood.
Bloodletting in hypertrophy, 74
  in dilatation, 87
Bloodletting in fatty degeneration, 106
  in valvular lesions, 250
  in acute pericarditis, 389
  in endocarditis, 449, 451
  in functional disorder, 480
  in thoracic aneurisms, 529
Blue disease, *vide* cyanosis.
Bouillaud on rupture of interventricular septum, 263
Bowditch on treatment of aneurisms, 534
Brain, symptoms referable to, in pericarditis, 352
Bruit de diable, 228
  de cuir neuf, 364
Bright's diseases in dilatation, 83
  in valvular lesions, 190
  in pericarditis, 336
  in endocarditis, 483
Bronchitis in valvular lesions, 178
Burrows on cerebral symptoms in pericarditis, 353
Budd on cirrhosis and valvular lesions, 187

C.

Carcinoma of heart, 124
Cathartics in pericarditis, 402
  in endocarditis, 450
  in valvular lesions, 255
Calcareous degeneration in aorta, 488
Carditis, 455
Cerebral symptoms in pericarditis, 352
Chambers on relative frequency of aortic and mitral lesions, 137
  on pericarditis and pyæmia, 341
  on pericardial adhesions, 114
Chamberlain, Dr. W. M., case of cavity opening into right ventricle, 242
Chevers on cyanosis, 271
  on pericardial adhesions, 419
Cheyne on respiratory movements in fatty degeneration, 101
Christison on external use of diuretics, 255
Cirrhosis of liver in valvular lesions, 187
Clark and Cammann, results of auscultatory percussion, 43
Cliquement metallique, 471
Clubbed fingers in cyanosis, 273
Coagula and clots in cavities of the heart, 275
  in cavities of the heart in endocarditis, 275
  in dilatation, 84
Coagula formed after death, 275
    during life, 276
  conditions of formation, 278
  symptoms of, 279
  physical signs of, 280
  diagnosis of, 281
  prognosis of, 284
  treatment of, 284
Collin, bruit de cuir neuf, 364
Complexion in valvular lesions, 192
Congenital malformations, *vide* malformations.
Congestion of lungs in valvular lesions, 171
  of liver, 185
  of kidneys, 189
  capillary, in valvular lesions, 192
Cor bovinum, 145
Cornea, fatty degeneration of, in cases of fatty degeneration of the heart, 101
Coronary arteries, obstruction of, in valvular lesions, 146
Cough in valvular lesions, 175, 252
  in pericarditis, 350
Counter-irritation in pericarditis, 395, 401, 403
  in endocarditis, 451, 452
Crisp on thoracic aneurisms, 508
Currents of blood, 135, 203
Current, direct mitral, 203
    aortic, 203
  regurgitant mitral, 204
    aortic, 204
Cyanosis in valvular lesions, 191
Cyanosis, 268
  anatomical conditions in, 268
  diagnosis of, 273
  prognosis in, 273
  treatment, 273
  in valvular lesions, 191
  in pericarditis, 349

D.

Da Costa, cases of simple hypertrophy, 31
Death, sudden, in valvular lesions, 257
  in fatty degeneration, 103
Decubitis in pericarditis, 352
Defects of heart, *vide* malformations.
Delirium, &c., in pericarditis, 353
  cases observed by author, 354
Dilatation, definition, and different forms of, 18
  mode of production, 79
  symptoms and effects of, 82
  physical signs of, 84
  summary of physical signs of, 87
  treatment of, 87
  of left ventricle from aortic regurgitation, 144
Digestion, disorder of in valvular lesions, 187

- Diastolic murmurs, 205
  - mitral direct murmur, 205
  - aortic regurgitant murmur, 212
- Diet in valvular lesions, 249
  - in acute pericarditis, 401
  - in endocarditis, 450, 452
- Digitalis in valvular lesions, 251
  - in hypertrophy, 78
  - in dilatation, 89
  - in fatty degeneration, 106
  - in pericarditis, 402
  - in functional disorder, 475
  - in thoracic aneurisms, 532
- Diuretics in cardiac dropsy, 254
  - external use of, 255
  - in pericarditis, 399, 402
- Dropsy in hypertrophy, 37
  - in dilatation, 83
  - in valvular lesions, 164, 191
  - treatment of, 253
- Dudley, Dr. Wm. H., case of simple hypertrophy of heart, 32
- Dyspnœa in hypertrophy, 38
  - in dilatation, 83
  - in fatty degeneration, 100
  - in valvular lesions, 172
  - in pericarditis, 349
  - in thoracic aneurisms, 511
- Dysphagia in pericarditis, 351
  - in thoracic aneurisms, 510
- Dyspepsia in functional disorder of heart, 463

E.

- Ectopia pectoralis cordis, 261
  - cordis ventralis, 261
    - cephalica, 261
- Eliminatives in pericarditis, 397, 402
  - in endocarditis, 450
- Emphysema of lungs, affecting the signs of enlargement, 46
  - a cause of enlargement, 28
  - in valvular lesions, 174
- Embolism, 168, 182, 436, 502
- Enlargement of the heart, 17
  - different forms of, 18
  - by hypertrophy and dilatation, 19, 22
  - physical signs of by percussion, 43
    - by palpation, 48
    - by auscultation, 58
    - by inspection, 68
    - by mensuration, 70
  - summary of physical signs of, 72
  - diagnosis of, 71
  - by dilatation, 79
    - mode of production, 80
    - symptoms and effects of, 80
    - physical signs of, 84
    - diagnosis of, 86
    - summary of physical signs of, 87
    - treatment of, 87
- Enterorrhœa in valvular lesions, 188
- Endocarditis, 424
  - anatomical characters of, 425
  - pathological relations and causation of, 431
  - artificial production of, 429
  - formation of coagula in, 436
  - symptoms of, 438
  - physical signs of, 440, 447
  - diagnosis of, 444
  - prognosis, 447
  - treatment of, 448
- Endo-pericarditis, 433
- Erythema in valvular lesions, 193
- Expectoration in valvular lesions, 175
- Exocardial sounds, *vide* friction-sounds.
- Exercise in hypertrophy, 77
  - in dilatation, 88
  - in fatty degeneration, 107
  - in valvular lesions, 249.
  - in functional disorder, 475
- Eyes, prominence of, 306
    - cases observed by author, 315
    - prognosis, 314
    - treatment, 319
  - contraction of in thoracic aneurisms, 515

F.

- Fatty degeneration, anatomical characters of, 94
    - from obstruction of coronary arteries, 98, 147
  - growth and degeneration, 94
    - anatomical characters of, 99
    - causes of, 97
    - symptoms and effects of, 99
    - physical signs and diagnosis of, 103
    - treatment of, 105
- Fevers, connection with pericarditis, 341
    - endocarditis, 435
- Fingers, bloodless in valvular lesions, 193
- Fibrin, remedies to promote coagulation of in thoracic aneurisms, 532
- Foramen ovale, patent, 264
- Forbes on angina pectoris, 299
- Friction-sounds in pericarditis, 364
  - mechanism of, 366
  - varieties of, 365
  - morbid conditions giving rise to, 366
  - distinguished from endocardial murmurs, 368
  - cardiac pleural, 371, 373
- Fremitus in thoracic aneurisms, 519
- Fuller on pericarditis in rheumatism, 337
  - on endocarditis, 431

G.

Gairdner on pericardial adhesions, 416
  on mechanism of dilatation of the heart, 81
  on contraction of pupil in aneurism, 515
Gangrene of lung in thoracic aneurisms, 516
Gastrorrhagia in valvular lesions, 188
Glottis, spasms of, in thoracic aneurisms, 508
Gout in functional disorder, 468
Graves on enlargement of the thyroid body, 307
Grisolle on coagula within the heart in various diseases, 278
  on polypi of heart, 285

H.

Hæmoptysis in hypertrophy, 37
  in valvular lesions, 175
Hallowell on rupture of heart, 121
Hartshorne, Dr. Henry, on muscular exhaustion of the heart, 464
Hemorrhage from bowels in valvular lesions, 188
  from nostrils in valvular lesions, 188
Heart, enlargement of, 17
  normal dimensions of, 20
    weight of, 22
  situation and anatomical relations of, 39
  physical signs of, 38
  apex-beat of, normal situation, 48
    in enlargement and hypertrophy, 50
  additional impulses of in enlargement, 52
  sounds of, *vide* sounds of heart.
  lesions affecting walls of, 91
  atrophy of, 91
  fatty growth and degeneration of, 94
  softening of, 109
  induration of, 114
  aneurism of, 115
  aneurism of coronary artery, 120
Heart, rupture of, 121
    in cardiac aneurism, 117
  lesions affecting valves and orifices of, 127
  congenital misplacements, malformations, and defects of, *vide* malformations.
  formation of coagula within, *vide* coagula.
  polypi of, *vide* polypi.
  inflammatory affections of, 328
  functional disorder of, 456
    varieties of, 457
Heart, functional disorder of, pathological relations and causation of, 459
    symptoms of, 57, 466
    physical signs of, 57, 466
    diagnosis of, 472
    prognosis of, 479
    treatment of, 480
Heart-sounds, abnormal modifications of in valvular lesions, 230
  reduplication of, *vide* reduplication of heart-sounds.
  abnormal modifications in pericarditis, 374
  in functional disorder, 470
Heart, rifle-ball in walls of, for twenty years, 125
  wounds of, 125
Hemorrhoids in bowels, 188
Hemiplegia in thoracic aneurisms, 513
Hope on suffering from dyspnœa in cardiac disease, 191
  on pericardial adhesions, 415
Hypertrophy, definition and different forms of, 18
  anatomical characters of, 23
  mode of production of, 25
  from aortic lesions, 26
  from mitral lesions, 27
  from tricuspid and pulmonic lesions, 28
  in pregnancy, 29
  from functional disorder, 30
  uncomplicated, 31
  concentric, 33
  symptoms and effects of, 35
  physical signs of, 38
  summary of physical signs of, 72
  treatment of, 74
  modifications of heart-sounds produced by, 65
  abnormal movements of præcordia in, 68
  diagnosis of, 71
  of left ventricle from aortic contraction, 144
Hydrothorax in valvular lesions, 179
Hysteria in functional disorder of the heart, 461
Hydatids in the heart, 123

I.

Impulse of heart, *vide* apex-beat.
  in epigastrium in health and disease, 55
  shock produced by, in health and disease, 56
  heaving in hypertrophy, 57
Impulses other than the apex-beat in health, 52
  in enlargement, 52
  in dilatation, 85

Inspection in enlargement of the heart, 69, 73
    in dilatation, 85
    in pericarditis, 377, 380
    in pericardial adhesions, 422
Induration of heart, 114
Iodine in chronic pericarditis, 409

K.

Karawagan on pericarditis in scurvy, 341
Kennedy on pericardial adhesions, 417
Kirkes on fibrinous deposits in arteries derived from the heart, 168
Kyber on pericarditis in scurvy, 341

L.

Lancisi on venous pulsation, 161
Law on anæmia of brain in valvular lesions, 183
Lesions of valves, *vide* valvular lesions.
    of aorta, *vide* aortic lesions.
    mitral, *vide* mitral lesions.
    tricuspid, *vide* tricuspid lesions.
    pulmonic, *vide* pulmonic lesions.
Liver, enlargement of, in dilatation, 83
        in valvular lesions, 186
    congestion of, in valvular lesions, 185
    cirrhosis of, in valvular lesions, 187
Lividity of face in valvular lesions, 192
    in pericarditis, 352
    in thoracic aneurisms, 511
Louis on softening of the heart in typhoid fever, 109
    on pericardial adhesions, 414
Lungs, congestion of, in valvular lesions, 171

M.

Malformations, congenital, 260
    varieties of, 261
    diagnosis of, 266
    causes of death in, 267
    treatment of, 267
Maculæ albidæ, 335
Mensuration in health, 70
    in enlargement of the heart, 71
    in pericarditis, 378, 380
Mental condition in valvular lesions, 184
    in functional disorder, 458, 474, 483, 485
    management of, in thoracic aneurisms, 531
Mercury in treatment of valvular lesions, 244
    in acute pericarditis, 392
Meigs on heart-clot, 276
Mitral valvular element of first sound of the heart, 68
    lesions, anatomical characters of, 138
    frequency of, 137
    water test in, 138
    effect on the heart-sounds, 135, 232
    diagnostic characters of, 235
Misplacements of heart, 260
Milk spots, 351
Mitral orifice, size of, 138
Morbus cœruleus, *vide* cyanosis.
Murmur, cardiac, in dilatation, 86
    mitral, direct or diastolic, 205
        regurgitant or systolic, 208, 228
    aortic, direct or systolic, 210
        regurgitant or diastolic, 212
    pre-diastolic, 213
    intra ventricular, 228
    hæmic, 226
    dynamic, 226
    in endocarditis, 441
    in thoracic aneurisms, 520
    tricuspid, direct, 240
Murmurs, endocardial or valvular, 195
    different qualities of, 196
    differences of pitch in, 197
    musical, 199
    exocardial or friction, 195
    organic and inorganic, 197, 200
    endocardial, abnormal conditions giving rise to, 200
        physical conditions involved in, 201
        dependence on valvular lesions, 202
        connection with obstructive and regurgitant lesions, 208
        distinguished from exocardial, 368
        pathological import of, 221
    produced within aorta, 213
    systolic and diastolic, 204
        localization of, 214, 221
    diastolic, localization of, 218
    inorganic, distinctive characters of, 226
    in functional disorder of the heart, 468
Myocarditis, 454

N.

Nervous system, symptoms referable to, in acute pericarditis, 358
    symptoms referable to, in functional disorder of the heart, 461
Nutmeg liver in valvular lesions, 186
Nutrition in valvular lesions, 185, 188

O.

- Œdema, pulmonary, in valvular lesions, 176 253n
- Opium in pericarditis, 395, 401
  - in endocarditis, 452
- Ormerod on pericarditis, 848

P.

- Palpation in enlargement of the heart and hypertrophy, 50
  - in dilatation, 85
  - in fatty growth and degeneration, 104
  - in pericarditis, 375, 380
  - in endocarditis, 440
  - in pericardial adhesions, 421
  - in functional disorder of the heart, 467
  - in thoracic aneurisms, 516
- Palpitation in valvular lesions, 148
  - in pericarditis, 347
  - in endocarditis, 439
- Paraplegia in thoracic aneurisms, 513
- Paracentesis of the pericardium, 409
- Parry on enlargement of the thyroid body, 306
- Paralysis in valvular lesions, 182
- Pain in valvular lesions, 147
  - in acute pericarditis, 345
  - in endocarditis, 438
  - in thoracic aneurisms, 513
- Peacock on malformations of heart, 268
  - on contraction and obliteration of the aorta, 493
- Percussion in enlargement of the heart and hypertrophy, 39
  - to determine boundaries of superficial and deep cardiac regions, 41
  - auscultatory, 43
  - as applied to discrimination of hypertrophy and dilatation and different portions of the heart enlarged, 44
  - in dilatation, 85
  - in fatty growth and degeneration, 104
  - in pericarditis, 360, 379
  - in pericardial adhesions, 421
  - in endocarditis, 440
  - in functional disorder of the heart, 467
  - in thoracic aneurisms, 519
- Pericardium, deficiency of, 262
  - adhesions of, 415
- Pericarditis, acute, anatomical characters of, 328, 329
    - stages of, 331
    - pathological relations and causation of, 336
    - symptoms of, 344
- Pericarditis, acute, symptoms of, referable to heart, 345
      - referable to circulation, 347
      - referable to respiratory system, 350
      - referable to digestive system, 351
      - referable to countenance, 352
      - referable to nervous system, 352
    - physical signs in, 360
    - percussion in, 360
    - auscultation in, 364
    - palpation in, 375
    - inspection in, 377
    - mensuration in, 378
    - summary of physical signs in, 379
    - diagnosis of, 380
    - prognosis in, 384
    - mode of dying in, 387
    - treatment of, 388
  - subacute and chronic, 404
    - anatomical characters of, 405
  - physical signs of, 407
  - treatment of, 409
- Pigment, atrophy of heart, 93
- Plethora in functional disorder of the heart, 460
- Pleuritis in valvular lesions, 179
  - in pericarditis, 339
  - in endocarditis, 434
- Pneumo-pericardium and pericarditis, 410
- Pneumonitis in valvular lesions, 179
  - in pericarditis, 340
  - in endocarditis, 434
  - formation of coagula within the cavities of the heart in, 275
- Portal congestion in valvular lesions, 185
- Polypi of the heart, 285
- Præcordia, enlargement of, &c., in enlargement of the heart, 68
- Pulse in hypertrophy, 35
  - in dilatation, 82
  - slowness of in fatty degeneration, 99
  - in valvular lesions, 150
  - in pericarditis, 347
  - in calcification of aorta, 491
  - in dilatation of aorta, 492
  - irregular and intermittent, 152
  - jerking, visible and deferred, 156
  - venous, 158
  - inequality in the two wrists and loss of in thoracic aneurism, 511
- Purring tremor, 233
- Purring tremor in functional disorder of the heart, 468
- Pulmonic orifice, normal size of, 139
  - artery, obliteration of, 264
- Purpura in pericarditis, 341

Pulmonic lesions, frequency of, 128
effect on the heart, 145
diagnostic characters of, 241

Q.

Quain on dependence of fatty degeneration on obstruction of coronary arteries, 147

R.

Recurrent laryngeal nerve, irritation of, and pressure on, in thoracic aneurisms, 507
Region, superficial cardiac, 40
deep cardiac, 40
Reduplication of heart-sounds, 320
mechanism of, 321
diagnosis of, 323
pathological import of, 327
Respiratory murmur in enlargement, 68
in pericarditis, 375
Respiration, aberration of in fatty degeneration, 101
Revulsives in pericarditis, 395, 401, 403
Rheumatism in relation to valvular lesions, 139
pericarditis in, 336
endocarditis in, 431
Risus sardonicus in pericarditis, 352
Richardson on the artificial production of endocarditis, 429, 435
Rogers, Dr. Stephen, case of thoracic aneurism, 514
Rupture of heart, 120, 117

S.

Scurvy in pericarditis, 341
Septa, ventricular and auricular, deficiency of, 264
Septum, supernumerary in ventricle, 264
interventricular, perforation of, cases, 225
Seidlitz on pericarditis in scurvy, 341
Sedatives in acute pericarditis, 394
Sibson on intensification of friction-sounds by pressure, 369
on capacity of pericardium, 332
on venous pulse in health, 161
on thoracic aneurisms, 500
Skoda on reinforcement of pulmonic sound, 232
Sleep in valvular lesions, 184
Snelling, Dr. F. G., addition to the binaural stethoscope, 59
Softening of heart, 109
of heart in fever, 112
of heart, treatment of, 113
Sounds of heart in health, 59
in hypertrophy, 65
in dilatation, 85
in fatty degeneration, 104
in softening, 113
in valvular lesions, 230
aneurismal, 521
Sound, first, or systolic, 59, 61
second, or diastolic, 59, 60
Sphygmoscope, 158
Sphygmographic characters of pulse in mitral insufficiency, 151
in mitral obstruction, 154
in aortic obstruction, 155
in aortic regurgitation, 157
Spleen, enlargement of in valvular lesions, 188
Stethoscope, binaural, advantages of, 59
Stokes on peculiar aberration of respiration in fatty degeneration, 101
on softening of the heart, 110
on enlargement of the thyroid body, 306
on affection of the voice in thoracic aneurisms, 509
on auscultatory signs in pneumo-pericarditis, 411
on aneurismal sounds, 520
Stillé on cyanosis, 269
Stimulants in pericarditis, 396, 401
Systolic murmurs, 204
mitral regurgitant murmur, 208
aortic direct murmur, 210

T.

Taylor on prominence of eyes, 306
on pericarditis in connection with pleurisy, 340
Testa on venous pulse, 161
Thyroid body, enlargement of, 306
cases observed by author, 315
Thoracic aneurisms, *vide* aneurisms, thoracic.
Thurman, cases of cardiac aneurism, 115
Tinnitus, 471
Tremor, purring, 233
Tricuspid valvular element in first sound of the heart, 63, 65
lesions, frequency of, 128
effect on heart, 145
diagnostical characters of, 239
orifice, normal size of, 139
regurgitation in health and disease, 146
direct murmur, 240
Tracheotomy in thoracic aneurisms, 508, 542
Tuberculosis of lungs in connection with pericarditis, 342
of heart, 123

Tuffnell's plan of the treatment of aneurisms, 583

U.

Undulation, quasi, in dilatation, 86
  in pericarditis, 378
  in pericardial adhesions, 422
Undefended space of interventricular septum, 268
Urine in valvular lesions, 189

V.

Valvular lesions, 127
  effects of on blood currents, 185
  pathological processes involved in production of, 139
  relations to rheumatism, 139
  symptoms of, referable to the heart, 142
  symptoms and pathological effects of, 141
    referable to the circulation, 164
    referable to the respiratory system, 171
    referable to the nervous system, 180
    referable to digestion and nutrition, 185
    referable to the genito-urinary system, 189
    referable to countenance and external appearance, 191
Valvular lesions, physical signs of, 194
  determination of the seat of, 214
  diagnostic characters of, 235
  treatment of, 244
  prognosis in, 245
Valvular lesions, innocuous, 136, 245
Valves, deficient and in excess, 265
Veins, pulsation and turgescence of, 159
Venous hum, 228, 469
Venous pulse, 158
Virchow on emboli, 168
Vocal resonance in præcordia as affected by enlargement of the heart, 68
Voice, auscultation of in pericarditis, 374
  in cases of thoracic aneurisms, 508
Vomiting in pericarditis, 351

W.

Weight of the heart, 22
White on external use of diuretics, 255
White spots on the heart, 351
Williams on treatment of iritis without mercury, 398
Woillez on comparison of the two sides of the chest by inspection, 69
Wounds of heart, 125

Z.

Zenker on softening of muscles, 110

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INDEX TO CATALOGUE.

	PAGE
Allen's Dissector and Practical Anatomist	6
American Journal of the Medical Sciences	1
Abstract, Half-Yearly, of the Med Sciences	3
Anatomical Atlas, by Smith and Horner	6
Ashton on the Rectum and Anus	28
Ashwell on Diseases of Females	22
Brinton on the Stomach	17
Bigelow on the Hip	30
Barclay's Medical Diagnosis	16
Barlow's Practice of Medicine	15
Bennet (Henry) on Diseases of the Uterus	22
Bowman's (John E.) Practical Chemistry	10
Bowman's (John E.) Medical Chemistry	10
Brande & Taylor's Chemistry	10
Brodie's Clinical Lectures on Surgery	28
Brown on the Surgical Diseases of Women	22
Buckler on Bronchitis	18
Bucknill and Tuke on Insanity	14
Budd on Diseases of the Liver	18
Bumstead on Venereal	19
Bumstead and Cullerier's Atlas of Venereal	19
Carpenter's Human Physiology	8
Carpenter's Comparative Physiology	8
Carpenter on the Microscope	8
Carpenter on the Use and Abuse of Alcohol	13
Carson's Synopsis of Materia Medica	13
Chambers on the Indigestions	17
Christison and Griffith's Dispensatory	13
Churchill's System of Midwifery	25
Churchill on Diseases of Females	22
Churchill on Puerperal Fever	22
Clymer on Fevers	17
Colombat de l'Isère on Females, by Meigs	22
Condie on Diseases of Children	21
Cooper's (B. B.) Lectures on Surgery	28
Cullerier's Atlas of Venereal Diseases	19
Curling on Diseases of the Testis	29
Cyclopedia of Practical Medicine	15
Dalton's Human Physiology	9
De Jongh on Cod-Liver Oil	13
Dewees's System of Midwifery	24
Dewees on Diseases of Females	22
Dewees on Diseases of Children	21
Dickson's Practice of Medicine	16
Druitt's Modern Surgery	28
Dunglison's Medical Dictionary	4
Dunglison's Human Physiology	9
Dunglison on New Remedies	10
Ellis's Medical Formulary, by Smith	13
Erichsen's System of Surgery	27
Erichsen on Nervous Injuries	27
Flint on Respiratory Organs	17
Flint on the Heart	17
Flint's Practice of Medicine	15
Fownes's Elementary Chemistry	11
Fuller on the Lungs, &c.	16
Gibson's Surgery	28
Gluge's Pathological Histology, by Leidy	14
Graham's Elements of Chemistry	10
Gray's Anatomy	6
Griffith's (R. E.) Universal Formulary	12
Griffith's (J. W.) Manual on the Blood, &c.	18
Gross on Urinary Organs	26
Gross on Foreign Bodies in Air-Passages	26
Gross's Principles and Practice of Surgery	26
Gross's Pathological Anatomy	14
Hartshorne's Essentials of Medicine	16
Hartshorne's Conspectus of the Medical Sciences	5
Hartshorne's Anatomy and Physiology	6
Habershon on Alimentary Canal	17
Hamilton on Dislocations and Fractures	28
Harrison on the Nervous System	14
Hoblyn's Medical Dictionary	4
Hodge on Women	23
Hodge's Obstetrics	24
Hodge's Practical Dissections	7
Holland's Medical Notes and Reflections	15
Horner's Anatomy and Histology	7
Hudson on Fevers,	18
Hill on Venereal Diseases	19
Hillier's Handbook of Skin Diseases	20
Jones and Sieveking's Pathological Anatomy	14
Jones (C. Handfield) on Nervous Disorders	18
Kirkes' Physiology	8
Knapp's Chemical Technology	11
Lea's *Superstition and Force*	31
Lea's Studies in Church History	31
Lallemand and Wilson on Spermatorrhœa	18
La Roche on Yellow Fever	14
La Roche on Pneumonia, &c.	14
Laurence and Moon's Ophthalmic Surgery	29
Lawson on the Eye	29
Laycock on Medical Observation	16
Lehmann's Physiological Chemistry, 2 vols.	9
Lehmann's Chemical Physiology	9
Ludlow's Manual of Examinations	5
Lyons on Fever	18
Maclise's Surgical Anatomy	7
Malgaigne's Operative Surgery, by Brittan	26
Marshall's Physiology	8
Markwick's Examination of Urine	18
Mayne's Dispensatory and Formulary	13
Mackenzie on Diseases of the Eye	28
Medical News and Library	2
Meigs's Obstetrics, the Science and the Art	25
Meigs's Lectures on Diseases of Women	23
Meigs on Puerperal Fever	23
Miller's System of Obstetrics	24
Miller's Practice of Surgery	27
Miller's Principles of Surgery	27
Montgomery on Pregnancy	24
Morland on Urinary Organs	28
Morland on Uræmia	18
Neill and Smith's Compendium of Med. Science	4
Neligan's Atlas of Diseases of the Skin	20
Neligan on Diseases of the Skin	20
Odling's Practical Chemistry	10
Pavy on Digestion	17
Prize Essays on Consumption	18
Parrish's Practical Pharmacy	11
Peaslee's Human Histology	7
Pirrie's System of Surgery	27
Pereira's Mat. Medica and Therapeutics, abridged	13
Quain and Sharpey's Anatomy, by Leidy	6
Ranking's Abstract	3
Roberts on Urinary Diseases	18
Ramsbotham on Parturition	25
Reese on Blood and Urine	18
Rigby on Female Diseases	22
Rigby's Midwifery	24
Rokitansky's Pathological Anatomy	14
Royle's Materia Medica and Therapeutics	13
Salter on Asthma	18
Swayne's Obstetric Aphorisms	25
Sargent's Minor Surgery	27
Sharpey and Quain's Anatomy, by Leidy	6
Simon's General Pathology	14
Simpson on Females	23
Skey's Operative Surgery	26
Slade on Diphtheria	18
Smith (J. L.) on Children	21
Smith (H. H.) and Horner's Anatomical Atlas	6
Smith (Edward) on Consumption	18
Smith on Wasting Diseases of Children	21
Solly on Anatomy and Diseases of the Brain	14
Stillé's Therapeutics	12
Tanner's Manual of Clinical Medicine	5
Tanner on Pregnancy	24
Taylor's Medical Jurisprudence	31
Thomas on Diseases of Females	22
Thompson on Urinary Organs	30
Thompson on Stricture	30
Todd and Bowman's Physiological Anatomy	9
Todd on Acute Diseases	17
Toynbee on the Ear	29
Wales on Surgical Operations	30
Walshe on the Heart	15
Watson's Practice of Physic	16
Wells on the Eye	29
West on Diseases of Females	23
West on Diseases of Children	21
West on Ulceration of Os Uteri	23
What to Observe in Medical Cases	16
Williams's Principles of Medicine	14
Wilson's Human Anatomy	7
Wilson's Dissector	7
Wilson on Diseases of the Skin	20
Wilson's Plates on Diseases of the Skin	20
Wilson's Handbook of Cutaneous Medicine	20
Wilson on Spermatorrhœa	18
Winslow on Brain and Mind	31

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